UNITED STATES DISTRICT COURT
DISTRICT OF MINNESOTA

In Bair Hugger: BAIR HUGGER FORCED AIR WARMING PRODUCTS LIABILITY LITIGATION	MDL No. 15-2666 (JNE/FLN)
This Document Relates To: *All Cases*	

MEMORANDUM IN OPPOSITION TO DEFENDANTS' MOTION TO EXCLUDE PLAINTIFFS' GENERAL CAUSATION MEDICAL EXPERTS

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INTRODUCTION

Operating room infections have long been a major concern for surgeons, particularly when performing orthopedic joint replacement procedures. These patients are especially susceptible to infection because implants have no blood supply and only a small inoculum of colony-forming units is needed to cause an infection. Because deep joint infections are both costly and life-threatening, infection control has been an important public health issue.

In recent decades, advances in operating room ventilation airflow technology significantly reduced infection rates by forcing airborne contaminants to settle to the floor of the operating room. But after Bair Hugger devices were commonly used, deep joint infection (DJI) rates increased. Though Defendants knew their product harbored dangerous pathogens and that its filtration system failed to remove most contaminants, they avoided conducting any studies to determine whether, and to what degree, Bair Hugger might increase the risk of deadly DJI. Instead, concerned scientists outside 3M conducted their own experiments. Mounting evidence from these studies confirmed that: 1) the Bair Hugger is a vector for microbial colonies; 2) the Bair Hugger's inadequate filtration system emits organisms into the surgical site; and 3) the Bair Hugger disrupts operating room airflow by blowing contaminants from the unsterile floor both upward and onto the patient.

The capstone of this pre-litigation research was the McGovern study, the first epidemiologic study to directly compare the risk of DJI in patients exposed to Bair Hugger versus other warming devices. Its results were alarming: Bair Hugger patients had nearly a four-fold increased risk of DJI compared to other patients. As is routinely done in peer-reviewed literature, the authors described the methods they used and their findings. The

article grew out of pre-litigation research, passed peer review, and was published in a respected medical journal; it has never been retracted. Nor have Defendants or any of their experts called for its retraction. There are no epidemiologic studies that contradict or disprove the association between Bair Hugger and DJI as reported in the McGovern study. The study is important because it not only provides a statistical measurement of DJI risk in patients exposed to Bair Hugger, but it also coheres with other experimental research.

The McGovern study angered Defendants—so much so that they hired two experts to do whatever they could to tear the study apart and reassemble it with contrived data, hoping to convince the Court that its findings are "statistically insignificant" and thus unimportant. Casting *Daubert* aside, Defendants invite the Court to commit reversible error by taking sides on the question of which party's experts are correct and resolving doubts in favor of excluding testimony. They also ask the Court to abuse its discretion by ruling that Plaintiffs' experts must reach their opinions to an absolute level of certainty—a demand the Eighth Circuit does not require. Using semantic sleights-of-hand, Defendants also call upon the Court to adopt a rule that the scientific community does not recognize—that experts cannot reliably base their opinions on studies finding only an "association," not "causation." This ruse flies in the face of standard scientific methods epidemiologists and other scientists trained in causation analysis routinely use in making causal inferences.

By placing an impossibly high standard on causation opinions, Defendants not only incorrectly redefine the standard of proof in tort law; they also improperly conflate admissibility of evidence and the weight to be accorded to it, usurping the role of the jury. Such an extreme view is antithetical to how science operates and is anomalistic under

Eighth Circuit law. The scientific evidence Plaintiffs' experts considered is valid, relevant, and admissible. That Defendants' experts drew different albeit erroneous conclusions from the evidence gives rise to a matter of weight for the jury, not an issue of admissibility for the Court to decide. The motion to exclude Plaintiffs' medical experts should be denied.

LEGAL STANDARDS UNDER *DAUBERT* AND EIGHTH CIRCUIT LAW

I. *Daubert* And Rule 702 Favor Admitting Expert Testimony

The Federal Rules of Evidence relax the traditional barriers to opinion testimony and thus favor admissibility. *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 587–88 (1993) (citing Fed. R. Evid. 401, 402, 702). Expert testimony is admissible under Rule 702 if: (1) the reasoning or methodology underlying the testimony is scientifically valid (reliable); and (2) the reasoning or methodology can properly be applied to the facts in issue (relevant). *Id.* at 592–93. The trial court must make an initial determination that expert testimony is both reliable and relevant before it may be admitted. *Id*. at 581, 597. Pertinent evidence based on scientifically valid principles easily satisfies those demands. *Id.* at 597.

Daubert suggested four non-exclusive factors to consider in assessing whether scientific evidence is reliable—none of which, alone, is determinative: (1) whether the scientific theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether a particular technique has a known potential rate of error; and (4) whether the theory or technique is accepted in the relevant scientific community. *Id.* at 593–94. An expert's reliance on peer-reviewed, published research meets at least the minimal criteria of good science. *Id.* at 593.

Scientific evidence is reliable if it is based on scientific methods. *Id.* at 590. The focus is on scientific principles and methodology, not conclusions. *Id.* at 595–96. It is unreasonable to require the subject of scientific testimony to be "known" to a certainty since science is an evolving process and "there are no certainties in science." *Id.* at 590.

Because experts often reasonably differ when analyzing scientific issues, the Supreme Court has instructed that the jury should resolve conflicting opinion testimony. *See Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 153 (1999); *see also Johnson v. Mead Johnson & Co., LLC*, 754 F.3d 557, 564 (8th Cir. 2014) (declaring that "the jury, not the trial court, should be the one to decide among the conflicting views of different experts").

Rule 702 codified this permissive approach in its 2000 amendment. *See* Fed. R. Evid. 702 advisory committee note (2000). As the Advisory Committee made clear, the exclusion of expert testimony "is the exception rather than the rule" and the district court's gatekeeper role "is not intended to serve as a replacement for the adversary system." *Id.*

II. Eighth Circuit Authority Follows *Daubert*'s Flexible Approach Regarding The Admissibility Of Expert Testimony

The Eighth Circuit has remained "consistently loyal" to the liberal standards of *Daubert* and Rule 702. *Lauzon v. Senco Prods., Inc.*, 270 F.3d 681, 687 n.2 (8th Cir. 2001). Indeed, "cases are legion that, correctly, under *Daubert*, call for the liberal admission of expert testimony." *Johnson*, 754 F.3d at 562; *accord Lauzon*, 270 F.3d at 686 (stating that Rule 702 is "one of admissibility rather than exclusion"). The "exclusion of an expert's opinion is proper only if it is so fundamentally unsupported that it can offer no assistance to the jury." *Wood v. Minnesota Mining and Mfg. Co.*, 112 F.3d 306, 309 (8th Cir. 1997).

The Eighth Circuit specifically instructs that proponents of expert testimony need not prove that the conclusions of their experts are correct. *Kuhn v. Wyeth, Inc.*, 686 F.3d 618, 625 (8th Cir. 2012). It also admonishes that trial courts are not empowered to determine which of several competing scientific theories has the best provenance. *Id.* at 633. A district court thus abuses its discretion if it resolves doubts in favor of excluding expert testimony or decides the correctness of an expert opinion. *Johnson*, 754 F.3d at 562.

As a general rule, "the factual basis of an expert opinion goes to the credibility of the testimony, not the admissibility." *Hose v. Chicago Northwestern Transp. Co.*, 70 F.3d 968, 974 (8th Cir. 1995). Rule 702 does not demand that a causation opinion be based on a "scientific absolute" to be admissible. *Adams v. Toyota Motor Corp.*, 867 F.3d 903, 916 (8th Cir. 2017). Flaws in an expert's methodology, or the novelty of scientific conclusions, do not warrant exclusion of expert testimony. *Bonner v. ISP Technologies, Inc.*, 259 F.3d 924, 929 (8th Cir. 2001); *Johnson*, 754 F.3d at 564. Such limitations go to the weight of the testimony. *Hose*, 70 F.3d at 974. Likewise, expert conclusions that have not yet been accepted by the scientific community are still admissible. *E.g.*, *Bonner*, 259 F.3d at 932.

As long as expert testimony rests on "good grounds, based on what is known," all disputes fall to the jury: "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence." *Daubert*, 509 U.S. at 596; *Johnson*, 754 F.3d at 562. In this Circuit, after all, the exclusion of expert testimony "is the exception rather than the rule." *See, e.g.*, *Robinson v. GEICO Gen. Ins. Co.*, 447 F.3d 1096, 1100 (8th Cir. 2006).

III. The Eighth Circuit Does Not Impose A "Scientifically Convincing" Standard For Medical Expert Testimony

In stark contrast to the liberal requirements of *Daubert* and its progeny, Defendants maintain that Eighth Circuit law calls for a higher standard of admissibility for medical expert testimony. *See* Defs.' Mem. at 5–7. Citing only *Glastetter*, an unsigned per curiam opinion, Defendants insist that medical expert testimony must be based on "scientifically convincing evidence" and not veer from "medical certainty." *See id.* (citing *Glastetter v. Novartis Pharms. Corp.*, 252 F.3d 986, 989 (8th Cir. 2001)). Defendants not only misstate the legal standard for medical expert testimony under *Daubert*; they misread *Glastetter*.

It is well established that *Daubert* applies equally to *all* types of experts—whether they are expressing opinions on scientific, medical, or other technical knowledge. *Kumho Tire Co.*, 526 U.S. at 149. The same flexible standard holds true under Rule 702, which admits opinion testimony with a reliable basis in scientific knowledge or experience, granting that latitude "to all experts, not just to 'scientific' ones." *Id.* at 148. As long as the testimony falls within the "range where experts might reasonably differ," it should not be excluded. *Id.* at 153; *Johnson*, 754 F.3d at 564. To demand that testimony derive from "convincing" or "certain" scientific evidence even where reasonable experts can interpret the evidence differently would impermissibly "set a separate, higher standard for scientists than for other witnesses with specialized knowledge." *In re Ephedra Prods. Liab. Litig.*, 393 F. Supp. 2d 181, 188 (S.D.N.Y. 2005) (discussing *Kumho Tire Co.*, 526 U.S. at 148).

Glastetter does not hold otherwise. There, the plaintiff's expert opined that the drug Parlodel could cause strokes based on the sole premise that it caused vasoconstriction, a

known cause of strokes. **But not a shred of direct evidence such as an epidemiologic study showing a statistical association between Parlodel and strokes supported the expert's unusual premise.** *Glastetter*, 252 F.3d at 990. Unsurprisingly, the Eighth Circuit therefore affirmed the exclusion of the plaintiff's expert because there was no reliable scientific evidence to support a showing of specific causation. *See id.* at 990–91; *see also Johnson*, 754 F.3d at 563 (explaining that expert testimony in *Glastetter* was excluded because the underlying evidence was "unreliable"); *In re Viagra Prods. Liab. Litig.*, 572 F. Supp. 2d 1071, 1081 (D. Minn. 2008) (concluding that *Glastetter* was not controlling because the data involved conditions that were "quite distinct" from the injury at issue). In so doing, *Glastetter* also disavowed the heightened standard now proposed by Defendants in this litigation, **explaining that the absence of definitive or direct evidence, such as observational studies, is not required to prove causation.** *Glastetter*, 252 F.3d at 992.

Neither the Eighth Circuit nor any other court within this jurisdiction has interpreted *Glastetter* as imposing any other restriction, let alone a heightened standard demanding "scientifically convincing" or "certain" evidence. Nor can Defendants cite such a case considering that every published decision here and elsewhere holds to the contrary. *See, e.g., In re Viagra Prods. Liab. Litig.*, 572 F. Supp. at 1081 (general causation testimony based on studies lacking "statistical significance" did not warrant exclusion of testimony); *In re Celexa and Lexapro Prods. Liab. Litig.*, 927 F. Supp. 2d 758, 764 (E.D. Mo. 2013) (concluding that "controversial" expert testimony on general causation did not affect admissibility). Properly understood, Defendants' gloss on *Glastetter* is not only contrary to Eighth Circuit law but it violates the liberal standards for admitting expert testimony.

IV. Plaintiffs' Experts Should Not Be Excluded For Relying On Research That Does Not Definitively "Prove" Causation

Inventing their own tautology to redefine epidemiology, Defendants also assert that the law forbids medical experts from relying on observational studies that do not "prove" causation or that "disclaim" causation. Defs.' Mem. at 8–9. In effect, Defendants conflate the statistical concept of "association" with the epidemiological concept of "causation"—a judgment scientists make after considering epidemiological data combined with *other* scientific evidence. This argument not only flies in the face of scientific methodology and Eighth Circuit legal standards, **but it contradicts the views of Defendants' own experts.** *See* PX1 (Holford Dep. at 375:12–17) (agreeing with the well-accepted proposition that "deciding whether associations are causal typically is not a matter of statistics alone, but also rests on scientific judgment"); PX2 (Borak Dep. at 67:24-68:3) (acknowledging the principle that "drawing causal inference after finding an association requires judgment").

By *definition*, epidemiologic studies can show only an association between exposure and outcomes. *See, e.g.*, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE, 3d ed. at 552, 598 (Federal Judicial Center 2011) ("An association is not equivalent to causation . . . Epidemiology cannot prove causation; rather causation is a judgment for epidemiologists and others."). Authors of epidemiologic studies therefore rarely if ever fully evaluate causation in their papers, "often calling for stronger evidence and more research before a conclusion of causation is drawn." *Id.* at 599. Contrary to Defendants' position, this does not mean causation does not exist; nor is it an expression of ignorance. It means that "all scientific fields are open-ended and can progress from their present state." *See id.* at n.143.

Evidence of an association from an epidemiologic study is the *starting* point for determining whether the reported association is causal. Once an association is reported, other scientists must explain or interpret it. *Id.* at 202. The Reference Manual explains:

> [C]ausation is a *judgment for epidemiologists* and others interpreting the epidemiologic data. Moreover, scientific determinations of causation are inherently tentative. The scientific enterprise must always remain open to reassessing the validity of past judgments as new evidence develops.
>
> In assessing causation, researchers first look for alternative explanations for the association, such as bias or confounding factors . . . Once this process is completed, researchers consider how guidelines for inferring causation from an association apply to the available evidence.

REFERENCE MANUAL at 598 (emphasis added).

One landmark set of guidelines epidemiologists use to make causal judgments is the Bradford Hill viewpoints. *Id.* at 599–600. Scientific and legal authorities have long endorsed these criteria as a reliable methodology. *Id.* The Hill viewpoints include multiple criteria; the most relevant ones here include temporal relationship, strength of association, biological plausibility, dose-response relationship, replication, consideration of alternative explanations, specificity of association, and consistency with other knowledge. *Id.* Courts in this jurisdiction and beyond have found the Hill viewpoints to be helpful tools for determining the reliability of expert testimony; however, failure to satisfy certain criteria is not grounds for exclusion. *E.g.*, *In re Viagra Prods. Liab. Litig.*, 572 F. Supp. 2d at 1081.

When applying these causation criteria, it is not uncommon for each party's medical experts to reach different causal conclusions based on the same set of scientific evidence.

"Even when competent, well-intentioned, conscientious scientists use identical data and identical generic considerations for interpreting it, they might reasonably and without mistake interpret the data and criteria somewhat differently." PX3 (C. Cranor, *Scientific Inferences in the Laboratory and the Law*, 95(S1) AM J PUBLIC HEALTH S124 (2005)). As other authorities explain, "[t]he fact that two scientists have different judgments about how much weight to give a study does not demonstrate that either has failed to use scientifically acceptable reasoning, but only that the ultimate opinion about the weight to accord a study is inherently part of the subjective judgment process of scientists." PX4 (Clapp R., *Environment and Health: Vital Intersection or Contested Territory?* AM. J. OF LAW & MEDICINE 30:189, 211 (2004)). Defendants' expert witnesses—most notably Professor Holford—agree. PX1 (Holford Dep. at 375:12–17) ("In the end, deciding whether associations are causal typically is not a matter of statistics alone, but also rests on scientific judgment."). For these reasons, the Eighth Circuit has always ruled that "although it is common that medical experts often disagree on diagnosis and causation, questions of conflicting evidence must be left for the jury's consideration." *E.g.*, *Hose*, 70 F.3d at 976.

The same is true when opposing experts interpret the same studies differently. *Id.* Several studies may contradict an expert's opinion, but it is not up to courts to choose between dueling theories. *Kuhn*, 686 F.3d at 633. In *Bonner*, for instance, the defendant faulted plaintiff's expert for relying on a study that reached a different conclusion than the expert. 259 F.3d at 932. The Eighth Circuit nonetheless upheld the admissibility of the expert's testimony, reasoning that the defendant's attacks on his opinion "indicate no more

than that his conclusion is not yet established as fact in the scientific community." *Id.* The validity of expert conclusions and their factual underpinnings are reserved for the jury. *Id.*

This Court followed suit in *In re Viagra Prods. Liab. Litig.*, where defendants sought to exclude the testimony of plaintiffs' causation expert because the epidemiologic studies he relied on lacked statistical significance, while other published studies directly contradicted his conclusions on causation. 572 F. Supp. 2d. at 1081–82. Judge Magnuson denied defendants' motion, finding that the published studies cited by plaintiffs' expert survived peer review, contained known rates of error, and were thus reliable. *Id.* The existence of contradicting studies did not undermine the experts' testimony because "questions of conflicting evidence must be left for the jury's determination." *Id.* at 1082.

Likewise, in the recent *Celexa* multidistrict litigation, defendants moved to exclude the plaintiffs' causation expert, arguing that he could not rely on a study that reached a contrary conclusion. *See In re Celexa and Lexapro Prods. Liab. Litig.*, 927 F. Supp. 2d at 765. Rejecting defendants' argument, the court reasoned that the plaintiffs' expert had adequately explained why he disagreed with the findings, further noting that "[e]xperts make these kinds of choices regularly in interpreting the data." *Id.* In the end, defendants were free to test any of the expert's underlying assumptions through cross-examination. *Id.*

Deliberately disregarding these decisions, Defendants instead rely on a cluster of cases from other circuits for the proposition that experts cannot infer causation from studies finding only an association. Defs.' Mem. at 8–9. This rigid approach turns the scientific method on its head. It is therefore hardly surprising that the vast majority of courts have

not adopted Defendants' attempt to undermine the permissive standard of Rule 702, while the few courts that have endorsed Defendants' position reflect a nonbinding minority view.

One such case is *McClain v. Metabolife*, 401 F.3d 1233 (11th Cir. 2005). There, the plaintiff's expert opined that ephedra, an herbal dietary supplement, could cause strokes. The expert based his opinion on several non-epidemiologic studies, case reports, and adverse event reports linking ephedra to sudden increases in blood pressure and stroke. Deconstructing each line of evidence, the court criticized the expert for relying on a paper in which the authors stated that their study did not prove causation. *Id.* at 1248. The court rebuked the expert's "willingness to exceed the limits of the conservative scientific methodology" employed by the authors in confining the conclusions to their research. *Id.*

McClain's holding not only ignores the clearly-established practice of applying statistical associations to existing knowledge in order to judge whether they are causal, *see* REFERENCE MANUAL at 589, but it vitiates the views of Defendants' experts. *See* PX1 (Holford Dep. at 375:12–17) ("In the end, deciding whether associations are causal typically is not a matter of statistics alone, but also rests on scientific judgment."). For this reason, courts across the country decline to follow *McClain*—and this Court should, too.

In *King v. Burlington N. Santa Fe Ry. Co.*, 762 N.W.2d 24 (Neb. 2009), for example, the Nebraska Supreme Court reversed the trial court's exclusion of expert testimony based on the unorthodox ruling in *McClain*, finding the trial court had clearly erred in applying a "conclusive study" standard when evaluating causation. *See id.* at 49. Consistent with Eighth Circuit law, the Nebraska Supreme Court declared that "individual epidemiological studies need not draw definitive conclusions on causation before experts can conclude that

an agent can cause a disease." *Id.* at 48. Where, as here, "the expert's methodology appears otherwise consistent . . . the court should admit the expert's opinion." *See id.* at 48–49.

The California Court of Appeal likewise disavowed *McClain*'s "conclusive study" rule in reversing the exclusion of plaintiff's causation opinion in *Davis v. Honeywell Int'l Inc.*, 199 Cal. Rptr. 3d 583 (Cal. App. 2016). Finding that the expert was entitled to draw conclusions from studies that did not prove causation, the court stressed that the gate for admissibility was not a "partisan checkpoint." *Id.* at 595. As in this Circuit, "[i]f the opinion is based on materials on which the expert may reasonably rely in forming the opinion, and flows in a reasoned chain of logic from those materials rather than from speculation or conjecture, the opinion may pass even though the court or the other side's medical experts disagree with its conclusion or the methods and materials used to reach it." *See id.*

Not long after *McClain*, a federal court overseeing the Ephedra MDL rejected the same "conclusive study" rule because it erroneously raised the plaintiffs' burden of proof:

> *Daubert* was designed to exclude "junk science." It was never intended to keep from the jury the kind of evidence scientists regularly rely on in forming opinions of causality simply because such evidence is not definitive. The legal standard, after all, is preponderance of the evidence, *i.e.*, more-probable-than-not, and that applies to causality as to any other element of a tort cause of action. Rule 702, a rule of threshold admissibility, should not be transformed into a rule for imposing a more exacting standard of causality than more-probable-than-not simply because scientific issues are involved.

In re Ephedra Prods. Liab. Litig., 393 F. Supp. 2d at 190.

Given the landslide of cases refuting the "conclusive study" rule in comparison to the scant authority supporting it,[1] no court in this jurisdiction has endorsed—much less adopted—this quaint rule. Scores upon scores of courts have even repudiated this "rule" as "wholly inconsistent with *Daubert* and the fundamental premise of Rule 702." *McClellan v. I-Flow Corp.*, 710 F. Supp. 2d 1092, 1101 (D. Or. 2010); *In re Heparin Prods. Liab. Litig.*, 803 F. Supp. 2d 712, 733–42 (N.D. Ohio 2011) (noting that reliance on studies that do not establish causation by a certainty do not render opinions "junk science"); *Monroe v. Zimmer U.S. Inc.*, 766 F. Supp. 2d 1012, 1027 (E.D. Cal. 2011) (noting that studies showing associations rather than proving causation may inform opinions) (citing *United States v. W.R. Grace*, 504 F.3d 745, 765 (9th Cir. 2007)). This Court should follow suit. *E.g.*, *Adams*, 867 F.3d at 916 (concluding that expert opinions regarding "causation need not be a 'scientific absolute in order to be admissible'") (citing *Bonner*, 259 F.3d at 929).

[1] For example, in a fractured decision, the Fifth Circuit excluded the testimony of plaintiff's expert because the articles he cited did not reach the same conclusion. *Huss v. Gayden*, 571 F.3d 442, 459 (5th Cir. 2009). The dissent decried the majority opinion as "dicta" that offered no helpful answers on scientific evidence, *id*. at 465–66, leading to criticism far and wide. Indeed, the Court of Claims reversed the lower court's reliance on *Huss* for exceeding the proper role of a gatekeeper and elevating the burden of proof. *Doe 93 v. Sec'y of Health & Human Servs.*, 98 Fed. Cl. 553, 569 n.13 (2011). The Fifth Circuit's decision in *Vargas v. Lee*, 317 F.3d 498 (5th Cir. 2003) has been criticized as well. *See, e.g., Kalantari v. Spirit Mountain Gaming, Inc.*, 5 Am. Tribal Law 94, 104 (Grand Ronde Tribal Ct. Mar. 24, 2004) ("[T]hose decisions narrow the gate too much when performing the Court's 'gatekeeping' function."). The remaining cases cited by Defendants are no more apposite. *E.g.*, *Newkirk v. ConAgra Foods, Inc.*, 727 F. Supp. 2d 1006, 1020 (E.D. Wash. 2010) (excluding expert since there was no "rhyme or reason" to his methodology).

V. Proof of "Background Rate" Is Not a Necessary Criterion for Reliable Expert Testimony On Causation

In addition to bungling Eighth Circuit law on the admissibility of expert testimony, Defendants erroneously contend that *Daubert* requires general causation experts to furnish evidence of "background risk" of disease in the general population. Defs.' Mem. at 9–10.

To the extent it even exists, this so-called "background risk" rule hails only from the Eleventh Circuit, where the *McClain* Court said that a "reliable methodology should take into account the background risk." 401 F.3d at 1243. The court cited no legal or scientific precedent to support this "rule" other than a primer on toxicology, which mentioned that "the likelihood that the chemical caused the disease or illness in an individual should be considered in the context of other known causes." *Id.* Based on this quotation—and this quotation alone—the court surmised that "other known causes" meant "the background risk of a specific disease—the risk that everyone faces of suffering the same malady that a plaintiff claims without having exposure to the same toxin." *Id.* But the article relied upon by the court contains no discussion of "background risk," and it further states that the analysis of "other known causes" does not necessarily apply to *general* causation.[2] PX5 (D. Eaton, *Scientific Judgement and Toxic Torts – A Primer in Toxicology for Judges and Lawyers*, 12(1) J LAW & POLICY 5, 40 (2003)) (emphasis in original).

[2] "Ecological" studies compare disease rates in the population of interest to a general background population. It is well-established that ecological studies provide weaker evidence than studies comparing disease rates in the more specific exposed versus unexposed groups—such as the McGovern study. *See* PX6 (A. Aschengrau, *Cohort Studies* at 193, ESSENTIALS OF EPIDEMIOLOGY IN PUBLIC HEALTH (Jones and Bartlett 2003)).

Defendants' citation to the REFERENCE MANUAL does not help their argument, for it likewise provides no support for excluding causation opinions that do not account for "background risk" of infection. *Cf.* Defs.' Mem. at 9. The REFERENCE MANUAL explains that epidemiologic studies such as the McGovern study are used to identify agents that may increase the risk of disease in populations and estimate the amount of excess risk these agents pose. *See* REFERENCE MANUAL at 552. By comparing groups who are exposed to an agent to those who are not, such studies determine whether there is an association between exposure and outcome. *Id*. at 218. Nowhere does the REFERENCE MANUAL instruct that data on "background risk" are the *sine quibus non* for finding an association or reaching an inference on causation; hence the reason that the Eighth Circuit along with nearly every other Circuit has not adopted Defendants' supposed "background risk" rule.[3]

Moreover, while "background risk" might be relevant in litigation involving toxic exposures and the like, it is not here. Defendants' own expert acknowledged, as he must, that "the use of intraoperative warming has become a standard of current surgical care." PX7 (Borak Rpt. at 3). The more germane question in this case is therefore whether Bair Hugger, as compared to other warming devices, increases the risk of deep joint infection—one of the very question answered by Plaintiffs' medical experts. *See* PX8 (Samet Rpt. at

[3] Claiming the "background risk" rule extends to "medical device litigation," Defendants rely on an unpublished opinion that excluded causation testimony because the expert failed to cite a study that "proved" causation. Defs.' Mem. at 10 (citing *Kilpatrick v. Breg*, 2009 WL 2058384, at *8 (S.D. Fla. June 25, 2009)). Courts have sharply criticized that court for veering beyond its gatekeeping role. *See Monroe*, 766 F. Supp. 2d at 1027; *McClellan*, 710 F. Supp. 2d at 1106; *Schott v. I-Flow Corp.*, 696 F. Supp. 2d 898, 905 n.3 (S.D. Ohio 2010).

4) ("the appropriate counterfactual is either no specific warming device or the use of a warming device that does not involve forced-air, the actual alternatives in practice").

And lest Defendants forget, **their own experts have disavowed the identical argument they raise here regarding "background risk."** Just consider the report of Dr. Borak, which unequivocally declares that "the hypothetical comparison of BH [Bair Hugger] vs. no warming device is *not relevant to* this dispute." PX7 (Borak Rpt. at 3) (emphasis added). Dr. Borak's report also asserts that "the alternative comparison, whether use of BH results in rates of SSI compared to use of non-FAW device, all other things being equal, *is the central question* here." *Id.* (emphasis added). These statements prove that Defendants will go to any measure—no matter the science, the law, or their own expert reports—to misguide this Court into erroneously excluding Plaintiffs' medical experts.[4]

ARGUMENT

An expert witness may testify based on scientific, technical, or other specialized knowledge if the court determines that: (1) the witness is qualified; (2) the testimony is relevant; and (3) the evidence supporting the expert's opinion is reliable. *Polski v. Quigley Corp.*, 538 F.3d 836, 839 (8th Cir. 2008). Because Plaintiffs' general causation experts easily meet each of these elements, Defendants spend little effort attacking their credentials or opinions on general causation. Instead, they take aim at the McGovern study itself, which provides direct epidemiological evidence of the association between Bair Hugger

[4] To the extent Defendants aver that "consideration of background risk" allows "experts to account for obvious alternative explanations," they put the cart before the horse in raising arguments on specific causation. Defs.' Mem. at 10. In any event, as noted *infra*, the admissions of Defendants' experts show that Plaintiffs have also proven specific causation.

and deep joint infection. As explained below, Defendants' arguments amount to nothing more than smoke and mirrors. Their conclusory attacks of the McGovern study not only lack scientific merit, but they lack credibility and border on bad faith argument. Drs. Samet, Jarvis, and Stonnington all base their causation opinions on a robust and coherent body of evidence. Under *Daubert*, their testimony is reliable, relevant, and therefore admissible.

I. Plaintiffs' Experts Are Well-Qualified Under Rule 702

Drs. Samet, Jarvis, and Stonnington are well respected and highly credentialed physicians in their respective fields of epidemiology, infectious disease, and orthopedic surgery. Defendants do not dispute their qualifications; nor ***can*** they, given the admissions of their own experts. *See* Defs.' Mem. at 10–13; *see, e.g.*, PX1 (Holford Dep. at 86:15–17) (admitting Dr. Samet is an expert in epidemiology). In developing their opinions, all three experts employed the sound methodology that every good scientist uses to reach a judgment on causation: they undertook a thorough review of the available published, peer-reviewed research relevant to the association between Bair Hugger and deep joint infections; and they applied their medical experience, expertise, and professional judgment in assessing the evidence. This approach bears the hallmark of reliable scientific technique.

A. Jonathan Samet, M.D., M.S.

Dr. Samet is a physician board certified in Internal Medicine and Pulmonology. He holds a Master's Degree in Epidemiology from the Harvard School of Public Health, where he also completed a three-year fellowship in clinical epidemiological research. *See* PX8 (Samet Rpt. at 1–2). He has served as Chair of Johns Hopkins Department of Epidemiology—one of the country's largest academic programs in infectious disease

methodology—teaching infectious disease epidemiology. *Id.* For nearly four decades, Dr. Samet has focused his research on the public health consequences of exposure to airborne particulates. *See id.* Dr. Samet has been broadly engaged in developing public policy related to indoor air quality and particulate matter, and his work involves the synthesis and evaluation of scientific evidence for causal inference and for risk assessment. *See id.* at 2.

Dr. Samet served as chair of the Committee on Research Priorities for Airborne Particulate Matter for the National Research Council. He has also assisted the Surgeon General in a series of landmark *causal inference evaluations*, **serving as the Senior Scientific Editor of the 2004 and 2014 Surgeon General Reports.** *Id.* at 2–3. As chair of the Clean Air Scientific Advisory Committee of the Environmental Protection Agency, Dr. Samet has guided the agency on evaluating evidence and inferring causation for pollutants, including airborne particulates. *Id.* at 3. He has published nearly 400 peer-reviewed papers, many of which address causation. PX9 (Samet Rpt. Ex. A at 37–69). Unlike Defendants' experts, Dr. Samet's opinions in this matter offer a multi-disciplinary perspective on the specific question of how scientists integrate epidemiological findings into the sum of relevant scientific evidence in making judgments on general causation. *Id.*

B. William Jarvis, M.D.

Dr. Jarvis is a physician board certified in Pediatrics with advanced training in Pediatric Infectious Diseases, Virology, and Epidemiology at Yale Medical School. He is globally renowned for his work on disease outbreaks. **From 1985-2002, Dr. Jarvis oversaw more infectious disease outbreaks than anyone else in the world.** For more than 20 years, he worked in the highest echelons at the Centers for Disease Control and

Prevention (CDC), specializing in healthcare-associated infections. In 2003, he was awarded the CDC Lifetime Scientific Achievement Award, and in 2010 he received the CDC's DHQP Lifetime Achievement Award in Epidemiology—bestowed once a decade.

Besides his work at the CDC, Dr. Jarvis served as a professor at Emory University Medical School and Emory's Rollins School of Public Health. He has published over 400 peer-reviewed publications and numerous papers in the areas of infectious diseases, infection control, epidemiology, and surgical site infections. Dr. Jarvis has extensive experience at the CDC in developing healthcare infection surveillance systems and conducting epidemiological studies and outbreak investigations. He has authored CDC guidelines for prevention of surgical site infections, and he helped develop the CDC's methodology for combining epidemiology and laboratory investigation to successfully investigate infectious disease outbreaks at hospitals. PX10 (Jarvis Rpt. Ex. A) (*passim*).

C. Michael Stonnington, M.D.

Dr. Stonnington is a physician board certified in orthopedic surgery. For the past two decades, he has performed total joint replacement and revision surgeries, along with complex trauma procedures. Dr. Stonnington has served as Chair of Surgery and Chief of Staff at Forrest General Hospital, a facility with over 400 physicians. He specializes in total joint replacement surgery and complex pelvic and acetabular surgery. He performs over 250 total joint replacement procedures per year. *See* PX11 (Stonnington Rpt.) (*passim*).

II. The Published and Peer-Reviewed McGovern Study Is A Valid Epidemiologic Study And Provides Important Evidence Of Causation

The medical experts for Plaintiffs and Defendants have different interpretations of the McGovern study. But the following facts are indisputable: It is a published, peer-reviewed epidemiologic study that found an association between Bair Hugger and DJI. Specifically, the study reported a statistically significant 3.8 fold increased risk of DJI from Bair Hugger compared to conductive warming blankets—the "alternatives" in practice. PX7 (Borak Rpt. at 3). As is routinely done in scientific papers, the authors described the methods they used and their findings. The study grew out of pre-litigation research, passed peer review, and was published in a respected medical journal; it has never been retracted. Nor have Defendants or any of their expert witnesses called for its retraction. There are no epidemiologic studies that contradict or disprove the association between Bair Hugger and DJI as reported in the McGovern study. Publication in a reputable peer-reviewed journal demonstrates that the study meets "at least the minimal criteria of good science." *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311, 1318 (9th Cir. 1995) ("*Daubert II*").

Epidemiologic studies have been well received by federal courts in mass tort suits, both in this district and nationwide. *E.g.*, *In re Viagra Prods. Liab. Litig.*, 572 F. Supp. 2d. at 1081. In the phenylpropanolamine (PPA) MDL, defense experts attacked a single epidemiologic study showing an association between PPA (decongestant) and hemorrhagic strokes—the Hemorrhagic Stroke Project (HSP)—based on a similar argument as Defendants raise here. *See In re Phenylpropanolamine (PPA) Prods. Liab. Litig.*, 289 F. Supp. 2d 1230, 1239 (W.D. Wash. 2003). They averred that a single observational study

could not show causation and that the HSP study had such serious flaws it was *per se* unreliable. *Id.* **The Honorable Barbara Rothstein, who now heads the Federal Judicial Center, disagreed and gave a name to this meretricious argument: "Defendants' *ex post facto* dissection of the HSP fails to undermine its reliability.** Scientific studies almost invariably contain flaws." *See id.* at 1240 (quoting REFERENCE MANUAL at 337).

Scientists widely recognize that all studies have limitations (or "flaws" as Defendants call them) that lead to uncertainty in how much weight to give the results. *See, e.g.*, REFERENCE MANUAL at 553. Scientists also understand that no single study can comprehensively address every potential confounding factor. *Id.* Despite their limitations, observational studies provide valuable information to the scientific community in estimating risks. For that reason, they are not only the mainstay of epidemiology but the primary form of evidence used to demonstrate causation inside the courtroom. *See id.* at 220 ("The bulk of the statistical studies seen in court are observational, not experimental."); *see also* PX12 (Nachtsheim Dep. at 325:22-328:13) (observational data is "valuable").

Legal authorities expressly recognize that the limitations of epidemiological studies do not generally detract from their reliability. *In re Viagra Prods. Liab. Litig.*, 572 F. Supp. 2d at 1081. As long as the study was peer-reviewed, published, contains known rates of error, and uses generally accepted methodology, it satisfies "the precise standards that the Supreme Court has signaled that the Court should use when determining reliability." *Id.*

III. At Best, All Of Defendants' Criticisms Of The McGovern Study Go To The Weight Of The Study, Not Admissibility

Though the McGovern study meets all the foregoing requirements and is not even necessary for Plaintiffs to prove causation, Defendants go to great lengths to disprove its findings. *See* Defs.' Mem. at 14–23; *cf. id.* at 24 ("epidemiological evidence is not absolutely necessary to support general causation expert opinions"). Yet a blizzard of red-herrings, misrepresentations, and flawed argument taint their pseudo-scientific analysis.

As previously explained, the unsurprising fact that the McGovern study "does not establish" causation or that its authors said the same does not detract from—much less doom—the opinions of Plaintiffs' medical experts. *See id.* at 15–16; *see also Glastetter*, 252 F.3d at 992. Dr. Samet, for example, explained that the McGovern study allowed him to "quantify the magnitude" of the risk pursuant to the 3.8 odds ratio reported in the study, but that he also relied on "different lines of evidence" in determining that Bair Hugger is a "substantial contributing cause" of DJI. *See* PX13 (Samet Dep. at 165:11-167:22); *see also* REFERENCE MANUAL at 568 ("The odds ratio ('OR') is similar to a relative risk in that it expresses in quantitative terms the association between exposure to an agent and a disease."). Defendants do not and cannot dispute that important distinction; in fact, they admit so in their briefing. Defs.' Mem. at 14 (recognizing that the study allows Plaintiffs' experts to "*quantify*[] the alleged increased infection risk from use of the Bair Hugger system") (emphasis added). 3M's broad attack on the McGovern study is thus misplaced.

Nor do Defendants' specific challenges to the study regarding purely hypothetical confounders and "tabulation errors" pass muster since they are built on one faulty premise

after another. *See id.* at 17–22. Defendants even rely on theories that flatly contradict the deposition testimony of their own "experts." *See id.* at 17–23. It stands to reason that Defendants' motion to exclude Plaintiffs' medical experts is not the product of good science. And even if it were, which it is not, its challenges go to weight, not admissibility.

A. Defendants Obfuscate the Deposition Testimony of the Study Authors.

Defendants initially cite but then distort the deposition testimony of the study authors. *See* Defs.' Mem. at 14–16. Indeed, before they even address the merits of the McGovern study, Defendants misrepresent the deposition testimony of Dr. Reed—one of the study authors. *Id.* at 14–15. Defendants maintain that Dr. Reed represented that Wansbeck Hospital "had been identified as a 'high outlier' in the English National Health System." *Id.* at 14. What they conceal from the Court, however, is more important. Dr. Reed also testified that not every hospital in the English National Health System reports as much infection data as Wansbeck Hospital; as a result, the survey data that Defendants' point to are known to be unrealistically "low." Pls.' Mem. to Exclude Holford at 17–18 (citing Reed Dep. at 67:9–15). Defendants' experts admitted as much in testifying that they could not determine or "know the degree of accuracy" of infection rates at Wansbeck. *Id.*

Even worse, Defendants cite Dr. Reed's deposition testimony for the phony proposition that "[t]here was no difference" in "whether the Bair Hugger system had a greater effect on operating room airflow" than conductive warming devices. *See* Defs.' Mem. at 15. **The transcript reveals no such statement.** To the contrary, the experimental portion of the McGovern study found a statistically significant difference in the number of particles that enter the sterile surgical field as a result of the Bair Hugger compared to

conductive warming devices. *See* PX14 (McGovern Study at 1540). **The study authors also testified repeatedly that they continue to stand behind their findings that Bair Hugger increases the risk of DJI.** *E.g.*, PX12 (Nachtsheim Dep. at 350:4–8); PX15 (Reed Dep. at 43:22–23); PX16 (McGovern Dep. at 415:8–20). And the scientists who peer-reviewed the study agree. *See* PX16 (McGovern Dep. at 375:9–14) ("This [study] demonstrates that there were actual changes in infection rates which would fit well with the experimental data and therefore support the contention that there is a serious issue to be addressed with [forced-air] warming devices."). Defendants' obfuscation of Dr. Reed's testimony along with the findings of the published McGovern study are thus misleading.

So, too, are the remaining citations to out of context snippets of deposition testimony that Defendants use to support their argument. *See* Defs.' Mem. at 15–16. For example, Defendants assert that Dr. Reed was "blunt in his deposition" that the McGovern study "doesn't establish causation," while they also cite Mr. Albrecht's testimony for the same reason. *See* Defs.' Mem. at 16. Try as they might to argue otherwise, **Defendants fail to explain that the authors fully endorsed the study's association of increased risk despite the fact that no observational study can *ipso facto* "prove" causation.** *See* PX16 (McGovern Dep. at 422:25-423:13); PX12 (Nachtsheim Dep. at 350:4–8); PX15 (Reed Dep. at 43:22–23); *see also* PX16 (McGovern Dep. at 377:7–8). This is the kind of standard cautionary language researchers commonly use when couching their conclusions. *See* REFERENCE MANUAL at 598–99 (authors of observational studies "often call[] for stronger evidence and more research before a conclusion of causation is drawn"). Professor Nachtsheim—a coauthor of the McGovern paper and statistics professor at the University

of Minnesota—confirmed this at his deposition, asserting that observational data are the "next best alternative" because they often "replicate randomized control[led] trials." *See* PX12 (Nachtsheim Dep. at 325:22-328:13) (noting that observational data is "valuable").

Despite their credentials to determine whether an association exists between Bair Hugger and DJI, the study authors (who are not trained epidemiologists or experts in causation analysis) did not cite to a methodology like the Bradford Hill criteria or attempt to do a comprehensive review of the evidence for drawing causal inferences. They did not address or opine on all the scientific evidence available in 2011 at the time they published the study, and of course they could not rely on the burgeoning body of scientific research since then supporting the nexus between airborne particles, bacteria, and DJI. *See, e.g.*, PX17 (Darouiche 2017). Ultimately, the fact that the McGovern authors did not undertake a causation analysis is par for the course in any research paper, but it in no way disqualifies some of the country's best scientists, including world-renowned epidemiologists such as Dr. Samet, infectious disease doctors such as Dr. Jarvis, and clinicians such as Dr. Stonnington, from rendering causal conclusions based on the their respective disciplines.

As Defendants' experts conceded at their depositions, causal determinations depend on the expertise and judgment of properly trained professionals, not absolute "proofs." *See* PX1 (Holford Dep. at 374:7-376:10). Defendants admit as much, noting that "[t]o "prove" 'cause' in a scientific sense would require a massive controlled, blinded study." Defs.' Mem. at 16 n.4. But Defendants have not only refused to [redacted]

[redacted]

[redacted][5] [redacted] Regardless, "proof by certainty" is not the correct legal standard, and the combination of peer-reviewed, published evidence of increased risk along with experimental studies showing the mechanism of injury easily satisfy the prerequisites for reliability and fit under *Daubert*.

In the same flawed vein, Defendants represent that the study authors "expressly acknowledge[d] several confounding variables." Defs.' Mem. at 17. The record shows this is patently false. While the McGovern study, as is routinely done in journal articles, noted its potential limitations—that the data "*may be* confounded by other inflectional control measures," the authors never, ever, "admitted" that these variables were, in fact, confounders and should be treated as such in the analysis. *Id.* The opposite is true. After the authors published the study, they conducted additional research that obviated those variables. *See* Pls.' Mem. to Exclude Holford at 25 (anti-thrombotic), 27–28 (antibiotic). **They also testified that the scientific literature on these factors provided no credible reason to treat these factors as confounders**. *Id.* **And their follow-up research found that the change in anti-thrombotic and antibiotic drugs were in fact *not* confounders**.

For example, based on one of these studies, which Defendants fail to cite, Dr. Reed unequivocally testified that "**we can [now] exclude Xarelto [the anti-thrombotic in the**

[5] [redacted]

McGovern study] as a confounding factor for infection rates."[6] PX15 (Reed Dep. at 215:14–18). Professor Nachtsheim, a professor of statistics at the University of Minnesota and Carlson School of Business, agreed with Dr. Reed. Given the great weight of scientific and statistical evidence, he reasoned, the change in anti-thrombotic during the McGovern study did not confound the drop in DJI rates.[7] *See* PX12 (Nachtsheim Dep. at 347:7–348:2).

The same holds true for antibiotics. Dr. Reed has not only stated that antibiotics have little to no effect in clearing up DJIs, *see* PX15 (Reed Dep. at 185:7–16), but he recently published a peer-reviewed paper finding **"no clear benefit to using one particular agent/regimen" or another for purposes of preventing DJI**, *see* PX21 (Reed 2015). Professor Nachtsheim also concluded that the change in antibiotic did not confound the findings of the McGovern study. *See* PX12 (Nachtsheim Dep. at 333:9–11). And while Defendants note that Mr. Albrecht testified that the "reduction in infections rates shown in the study could be due to the adoption of conductive fabric or it could be due to outside factors," *see* Defs.' Mem. at 17, he did not know one way or another; nor was he aware of all the peer-reviewed and published studies that soundly put Defendants' argument to bed.[8]

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[6] Dr. Reed's opinion deserves just as much weight as Defendants' experts considering that defense counsel touts Mr. Reed as a "prominent researcher[] in the U.K. [who has] received funding from 3M for important research activities over the years." PX49 (Sept. 19, 2016 Letter from Mr. Jerry Blackwell). Indeed, according to defense counsel, "Dr. Reed is currently involved in an unrelated research project for another group within 3M." *See id.*

[7] Plaintiffs have not retained Professor Nachtsheim as an expert witness in this litigation, but his opinion is entitled to the same weight because Defendants' experts admitted that he was an "expert in the field of statistics." *See, e.g.*, PX1 (Holford Dep. at 87:8–11).

[8] *See* Pls.' Mem. to Exclude Holford at 22–30; *see also* PX22 (B. Eriksson, *Rivaroxaban versus Enoxaparin for Thromboprophylaxis after Hip Arthroplasty*, 358(26) N. ENGL. J.

B. Professor Holford's Testimony Hurts, Not Helps, Defendants.

Stymied by the results of a reliable peer-reviewed study, Defendants turned to Professor Holford to mix and match and ultimately "recalculate" data sets in order to support Defendants' speculative theory that certain variables might have confounded the McGovern study. *See* Defs.' Mem. at 19. But Professor Holford collapsed on cross-examination, **conceding that the scientific literature does not suggest a relationship between anti-thrombotic drugs and DJI**. *See, e.g.*, PX1 (Holford Dep. at 293:7-295:13). Although he failed to offer any good reason for ignoring every study that has disclaimed an association between anti-thrombotic drugs and DJI, *see* Pls.' Mem. to Exclude Holford at 21–25 (collecting peer-reviewed studies showing no significant relationship between DJI and anti-thrombotic drugs), he did agree that his attempt to control the anti-thrombotic drug in the McGovern study was **"not a very good estimate."** PX1 (Holford Dep. at 219:8–23). **He also admitted that his risk estimate was two times *more variable* than the risk ratio reported in the McGovern study.** *See id.* at 220:7–10. Without an *a priori* basis to

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MED. (2008)); PX23 (A. Kakkar, *Extended Duration Rivaroxaban versus Short-Term Enoxaparin for the Prevention of Venous Thromboembolism after Total Hip Arthroplasty*, 372 LANCET 31-39 (2008)); PX24 (M. Lassen, *Rivaroxaban versus Enoxaparin for Thromboprophylaxis after Total Knee Arthroplasty*, 358(26) N. ENG. J. MED. (2008)); PX25 (A. Turpie, *Rivaroxaban versus Enoxaparin for Thromboprophylaxis after Total Knee Arthroplasty (RECORD 4)*, 373 LANCET 1673-80 (2009)); PX26 (C. Jensen, *Return to theatre following total hip and knee replacement, before and after the introduction of rivaroxaban*, 93-B(1) J BONE & JOINT SURGERY Br., 93-B, 1 (2011)); PX27 (S. Jameson, *Wound Complications Following Rivaroxaban Administration*, 94 J BO E JOINT SURG. AM. 1554-8 (2012)); PX21 (C. Hickson, *Prophylactic Antibiotics in Elective Hip and Knee Arthroplasty*, 4 BONE JOINT RES. 101, 186 (2015)); PX28 (A. Melling, *Effects of Preoperative Warming on the Incidence of Wound Infection After Clean Surgery*, 358 LANCET 876, 876 (2001)).

determine that the change in anti-thrombotic confounded the McGovern study, Defendants' experts have fabricated a connection for purposes of this case *sui generis*. *See id.* at 26. But science, unlike patient warming devices, cannot be so cheaply manufactured.

Professor Holford's *ipse dixit* regarding the change in antibiotic falls victim to the same mistakes. *See* Defs.' Mem. at 19. In addition to the testimony of the study authors, the scientific literature finds "no clear benefit to using one particular antibiotic agent" versus another for purposes of preventing DJI. *Id.* at 27–28. Professor Holford, moreover, admitted on cross-examination that the change in antibiotic from gentamicin to gentamicin plus teicoplanin actually ***reduced*** **deep joint infection rates among patients who used the *Bair Hugger* while it *increased* infection rates among patients who used *conductive warming devices*.** *Id.* at 28 (citing Holford Dep. at 317:2-322:14). **If anything, then, his calculations show that the increased risk of DJI from the Bair Hugger may be even *greater* than the 3.8 odds ratio reported in the McGovern study.** *See* PX29 (Holford Rpt. at 6) (calculating higher infection rates from using protocol two versus protocol one).

Professor Holford's testimony not only supports a showing of general causation in this regard, **but it actually *proves specific causation* by itself**. On cross-examination, Plaintiffs' counsel asked Professor Holford whether he agreed with the following: "If the incidence of disease in an exposed group is more than twice the incidence in the unexposed group [i.e., risk ratio > 2.0], the probability that exposure to the agent caused [the same disease] in a similarly situated individual is also greater than 50%." *See* PX1 (Holford Dep. at 225:19–226:1). He unhesitatingly agreed—and for good reason. *Id.* Numerous courts, including this one, have recognized "that a RR [risk ratio] of 2.0 or greater provides *reliable*

evidence of specific causation." *See In re Viagra Prods. Liab. Litig.*, 572 F. Supp. 2d at 1078 (emphasis added) (citing the REFERENCE MANUAL ON SCIENTIFIC EVIDENCE at 384).

In *Manko v. United States*, 636 F. Supp. 1419, 1438 (W.D. Mo. 1986), the trial court offered the following explanation that mirrors Professor Holford's testimony in this case:

> A relative risk of "2" means that the disease occurs among the population subject to the event under investigation twice as frequently as the disease occurs among the population not subject to the event under investigation. Phrased another way, a relative risk of "2" means that, on the average, there is a fifty percent likelihood that a particular case of the disease was caused by the event under investigation and a fifty percent likelihood that the disease was caused by chance alone. A relative risk greater than "2" means that the disease more likely than not was caused by the event.

The Eighth Circuit not only upheld the decision on appeal, *see Manko v. United States*, 830 F.2d 831 (8th Cir. 1987), but myriad courts since then have concluded that an odds ratio greater than two is sufficient to prove specific causation. *E.g.*, *Liable v. Sec'y of Health & Human Servs.*, 2000 WL 1517672, at *15 (Fed. Cl. Sept. 7, 2000) (collecting appellate cases and stating that a "relative risk estimate of 3.3 means that it is statistically reasonable to point to an *individual case* and to say that it is 'more likely than not' that such *individual's* chronic dysfunction was vaccine-caused") (emphasis added); *cf.* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 3d. at 616 ("[P]laintiff may satisfy his or her burden of production even if a relative risk less than 2.0 emerges from the epidemiologic evidence.").

As in those cases, this case involves an observational study reporting a risk ratio over two. But unlike in those cases, the McGovern study reports an even higher risk ratio of nearly four, while data collected after the publication of study continued to show a risk

ratio of 3.6. *See* PX16 (McGovern Dep. at 414:6–12) ("This data shows there is a 3.6 times increase in infection as a result of using forced-air warming devices compared to conductive-fabric warming devices."); *see also* PX8 (Samet Rpt. at 11–12) (3.6 "risk ratio" very similar to 3.8 "odds ratio"). As Dr. McGovern explained at his deposition, "both this odds ratio and the odds ratio reported in the final published McGovern study are above 3.0," meaning that orthopedic patients who are warmed by Bair Hugger have a "three times or more higher incidence of infection." *See* PX16 (McGovern Dep. at 414:14-415:20).

What's more, Professor Holford admitted on cross-examination that even when he controlled for his nonexistent confounding factors, such as the change in anti-thrombotic drugs, **his calculations *still* showed a "doubling of the risk."** PX1 (Holford Dep. at 291:7–21). Compared to most cases involving epidemiologic studies, Professor Holford's deposition testimony shows that Plaintiffs have already proven specific causation *a fortiori*.

Given Professor Holford's helpful expert testimony, Defendants revert to misstating the meaning of Mr. Albrecht's fact testimony. According to Defendants, Mr. Albrecht admitted there would not be a significant difference in DJI rates if the antibiotic and anti-thrombotic drugs were "accounted for" in the McGovern study. *See* Defs.' Mem. at 18–19.

To be sure, Mr. Albrecht stated that the infection rates were similar when both variables were controlled; read in context, however, he testified that there were "not enough infections" in such a small population to generate "properly powered" results. *See* PX30 (Albrecht Dep. at 217:13-218:4) ("This data, there's possibly not enough infections – infections to do a multivariate analysis like that where it's properly powered."). Defendants ignore these elementary scientific and statistical principles in representing Mr. Albrecht's

testimony for what it is not, even though their internal documents reveal the opposite. *See* REFERENCE MANUAL at 254 ("Discerning subtle differences requires large sample sizes; small sample sizes may fail to detect substantial differences."); [redacted]

[redacted] **Professor Holford stated as much at his deposition, confessing that his attempt to control the anti-thrombotic and antibiotic created twice as much unpredictability as the study he tried to attack**. *See* PX1 (Holford Dep. at 325:8-326:1).

Precisely for these reasons, Dr. McGovern and Professor Nachtsheim testified under oath that neither the anti-thrombotic nor the antibiotic should have been controlled. *See* PX12 (Nachtsheim Dep. at 340:5–11, 349:14–25); PX16 (McGovern Dep. at 385:4–14) (finding no reason to "deselect patients from the population presented in this study for those who received a different type of antibiotic than others"). Defendants' expert, Professor Holford, also stated that controlling both variables led to shaky conclusions and that any attempt to control for non-confounding variables created unnecessary and unreliable "variance." *See* Pls.' Mem. to Exclude Holford at 29 (citing Holford Dep. at 325:8-326:1).

Unfortunately for Plaintiffs and the Court, not even Defendants' "experts" can stop them from making unscientific arguments. Yet Defendants still move to exclude Plaintiffs' experts with the hyperbolic and ultimately misleading argument that the McGovern study does not "show an association between Bair Hugger use and increased infections, once even one of its several confounding factors . . . is accounted for." *E.g.*, Defs.' Mem. at 2.

C. Defendants and Their Experts "Manufacture" Tabulation Errors.

In addition to obfuscating the testimony of the authors, Defendants rely on Professor Holford's report to assert that "undisputed" tabulation errors exist in the McGovern study. *See* Defs.' Mem. at 19. But in contrast to his report, Professor Holford's sworn testimony reveals that he did not use the final data of the McGovern study to determine whether it suffered from "major tabulation errors." *See* Pls. Mot. to Exclude Holford at 9–11. Failing to conduct any independent research, he instead relied on a draft data set that he received from 3M—among only 19 other documents—and accepted the data at face value. *Id.* at 9.

Given the dearth of his knowledge on the subject, much less the McGovern study itself, Professor Holford conceded that he did not know whether the data he relied upon for purposes of his testimony were the same data the authors used in the study. *See id.* at 9–10 (admitting that the data he relied on contradicted the data depicted in Figure 7 of the study). Consider the following colloquy between Plaintiffs' counsel and Professor Holford:

> Q: Mr. Gordon [3M's counsel] doesn't know if Exhibit 10 is the original data set.
>
> A: Okay.
>
> Q: Mr. Albrecht [coauthor of the McGovern study] doesn't know whether Exhibit 10 is the original data set.
>
> A: Yeah.
>
> Q: Mr. Borak [Defendants' medical expert on general causation] also doesn't know whether Exhibit 10 is the original data set.
>
> A: Okay.
>
> **Q: And you don't know.**

A: I don't.

See PX1 (Holford Dep. at 128:21-129:6) (emphasis added).

At best, Professor Holford only knew that the draft data set he used in all his calculations was incomplete and different from the data the authors used in the published and peer-reviewed McGovern paper. *See, e.g.*, Pls.' Mem. to Exclude Holford at 9. These admissions defeat Professor Holford's remixed recalculations along with Dr. Borak's expert report, which wholly relied on Professor Holford's attempt to mix and match variables without conducting any independent analysis.[9] *See id.* at 11 (compiling deposition testimony from authors stating there were no tabulation errors); Pls.' Mot. to Exclude Borak at 7 (noting Dr. Borak relied on Professor Holford).[10] Plaintiffs' medical experts were thus under no obligation whatsoever to "correct" such "errors." Defs.' Mem. at 20.

To the extent Defendants cite Dr. Samet's deposition testimony for the erroneous proposition that Professor Holford "did the calculations correctly," 3M ignores the forest for the trees. *See* Defs.' Mem. at 19 (emphasis added). In making that specific averment, Dr. Samet was not referring to the flawed inputs, data, or statistical methods underlying

[9] Even though Professor Holford concocted Defendants' pseudo-scientific theory of a "tabulation error," Defendants curiously attempt to attribute the genesis of the theory to Dr. Borak, thereby quietly distancing themselves from Professor's Holford blunder. Defs.' Mem. at 19 ("In his expert report, Dr. Jonathan Borak . . . notes that one of the HotDog infections was tabulated as a Bair Hugger infection.") (citing Holford Rpt. at 2–3 & n.1).

[10] For these reasons, along with the arguments stated in Plaintiffs' motion to exclude Professor Holford, 3M's reliance on *In re Viagra Prod. Litig.*, 658 F. Supp. 2d 936, 944–45 (D. Minn. 2009), is misplaced. *See* Defs.' Mem. at 19 (citing case for the proposition that study was not reliable because the "plaintiffs failed to rebut miscodings and errors").

Professor Holford's remixed calculations. The full deposition transcript unveils that he was only referring to the single calculation in "Footnote 1" of Dr. Holford's expert report. *See* PX13 (Samet Dep. at 125:23-126:7) (asking whether Dr. Samet "had any reason to think that Professor Holford screwed up the calculations that he did there") (emphasis added).

Along with ignoring the context of Dr. Samet's testimony, Defendants blithely disregard what Professor Holford actually found in "Footnote 1" of his report. **His calculations not only show a significant relationship between use of Bair Hugger and DJI (p=.0356), but his calculations still show at least a doubling of the risk of DJI from using Bair Hugger in orthopedic surgeries (OR=2.86) even assuming, *arguendo*, that the McGovern study contained a so-called "tabulation error."**[11] *See* PX29 (Holford Rpt. at 3 n.1). Dr. Reed explained as much at his deposition, noting that one more or less infection did not impact the scientific or clinical impact of the study, despite Defendants' argument to the contrary. *See* Pls.' Mem. to Exclude Holford at 11 (adding or subtracting one DJI from the total number of infections does not impact "scientific, human, or economic" significance of the findings of the McGovern study); *cf.* Defs.' Mem. at 19.

Finally, citing Dr. Borak's report in yet another attempt to misguide the Court, Defendants contend that "infection data from an eight-month period was improperly excluded" from the McGovern study. *See* Defs.' Mem. at 19. Had the study not excluded "eligible SSI data from 10/07 to 6/08," say Defendants, "the study would have had no significant clinical findings." *Id.* This argument falls apart for two straightforward reasons.

[11] Even under the worst case scenario, then, Professor Holford's calculation still support a finding of both general *and* specific causation. *See, e.g.*, *Liable*, 2000 WL 1517672, at *15.

First, Dr. Reed made clear at his deposition that "full-time surveillance" did not start until July 1, 2008, immediately *after* the date fabricated by Defendants and their experts. *See* Pls.' Mot. to Exclude Holford at 20. Indeed, on cross-examination, Professor Holford conceded that he had no reason to doubt that full-time surveillance began on July 1, 2008 instead of October 8, 2007. *Id.* at 20–21. Second, the absence of statistical significance does not mean an absence of clinical significance. Defendants' experts admitted so on-cross examination—as they must, given the treatment of this issue in standard epidemiological treatises.[12] *See id.* at 14–16. Defendants' assertions thus do not hold water.

D. Defendants Only Speculate About "Undisclosed" Confounders.

Failing to demonstrate, much less prove, that any "disclosed" variables confounded 3.8 odds ratio reported in the McGovern study, Defendants fall back on the perplexing argument that certain "undisclosed" variables confounded the study. *See* Defs.' Mem. at 20–21. Besides citing an online newsletter authored by a nurse without any training in epidemiology or other scientific methods for making causal determinations, Defendants fail to cite a scintilla of scientific evidence to support their novel argument. Nor can they.

As discussed in Plaintiffs' motions to exclude the testimony of Professor Holford and Dr. Borak, these immaterial variables were not identified in the McGovern study for a simple reason: while they *might* impact surgical site infections, they have *no known impact* on the outcome of interest in this litigation—deep joint infections. *See* Pls.' Mot. to Exclude

[12] In any event, Defendants' own expert confessed that even when applying the most conservative statistical test, the results of the McGovern study were still significant. PX1 (Holford Dep. at 175:23-176:15) (finding significance under Fisher's test or chi-squared).

Holford at 22–23; *see also* Pls.' Mot. to Exclude Borak at 9–10, 18–20. **Given this undisputed fact, Dr. McGovern explained that the scientific literature shows that none of these changes significantly impact DJI rates**, PX16 (McGovern Dep. at 408:17-409:25), **and Professor Holford readily agrees**, PX1 (Holford Dep. at 306:17-308:14).

Unsurprisingly, neither Defendants nor their experts have cited a single peer-reviewed study showing that any of these "undisclosed" variables significantly reduce surgical site infections, much less DJI. *See* PX1 (Holford Dep. at 305:13-306:10) (admitting deep joint infections are "not the same thing" as surgical site infections). Indeed, each and every one of the eleven "SSI" interventions listed in Defendants' briefing focuses on "skin-preparation" and other topical measures that have absolutely nothing to do with infections that occur on hip or knee implants, which, of course, are placed *under* the patient's skin and subcutaneous tissue. *See* Defs.' Mem. at 21. Defendants cannot fabricate science to suit their legal arguments; nor can they reasonably represent that Wansbeck Hospital had "out-of-control orthopedic infection rates" when their internal documents

[redacted]

In short, Defendants' reliance on a bouillon of "undisclosed" yet unproven confounders amounts to nothing more than sheer sophistry. If they were correct, *all* medical expert testimony derived from observational and other epidemiologic studies would be inadmissible. That is not—and cannot be—the law. *See, e.g.*, *Johnson* 754 F.3d at 564 (concluding that expert's incomplete testing and failure to consider potential confounding factors did not undermine the reliability of his testimony; rather, it was up to the jury to

evaluate the veracity of his conclusions); *see also* REFERENCE MANUAL at 553 (recognizing that no study alone can comprehensively address every potential confounder).

Accordingly, each and every one of Defendants' arguments against the McGovern study fall flat; and even assuming, for the sake of argument, the Court entertains those arguments, they are the essence of disputes that are reserved for the jury, not the Court. *See, e.g.*, *Johnson*, 754 F.3d at 564 ("the jury, not the trial court, should be the one to "decide among the conflicting views of different experts") (citing *Kumho Tire Co.*, 526 U.S. at 153); *see also Hill v. Southwest Energy Co.*, 858 F.3d 481, 486 (8th Cir. 2017) (holding that court abused its discretion in excluding plaintiff's expert whose calculations and opinion testimony were "imperfect" but "reliable enough to assist the trier of fact").

IV. Plaintiffs' Experts Also Rely On Non-Epidemiologic Studies Which Provide Compelling Support For Causation When Combined With McGovern et al.

Recognizing the futility of challenging the McGovern study, Defendants shift gears and target the great weight of experimental studies documenting how Bair Hugger transports particles and bacteria to the surgical site. Defs.' Mem. at 24–28. As explained above, scientific and legal authorities agree that causal inference requires the evaluation of scientific evidence from epidemiologic studies, laboratory experiments, and any other relevant information. REFERENCE MANUAL at 23, 553. Thus, even when an observational study finds "only" an association, Plaintiffs' medical experts must not be precluded from testifying "if they adequately explain why the association is valid and how causation can be inferred from it." *McClellan*, 710 F. Supp. 2d at 1102. The Bradford Hill viewpoints are

a generally accepted methodology used by scientists for drawing causal inferences based on the available evidence. *See* REFERENCE MANUAL at 600; *see also* PX4 (Clapp at 199).

Applying this methodology, Plaintiffs' experts considered not only the McGovern study, but a panoply of published, peer-reviewed experimental studies. These experimental studies, by themselves, were neither designed nor intended to measure the statistical association between Bair Hugger and DJI—the McGovern study already does that. Rather, the purpose of these non-epidemiologic studies is to describe the *mechanisms* by which the Bair Hugger transmits contaminated airborne particles onto the sterile surgical site during orthopedic surgeries.[13] *See* PX32 (Bradford Hill, *The Environment and Disease: Association or Causation?* 58 PROC. ROYAL SOC'Y MED. 295, 298 (1965)). Individually, these peer-reviewed studies are concordant with one another; collectively, they provide a coherent and compelling explanation for the strong association between Bair Hugger and DJI as reported in the McGovern study. *Id.* From the totality of this scientific information, Plaintiffs' experts reliably concluded that Bair Hugger can cause DJI in orthopedic patients.

Violating the Bradford Hill methodology, Defendants argue that none of these experimental studies concludes that Bair Hugger "causes" DJI, so Plaintiffs' medical experts may not consider them. *See* Defs.' Mem. at 24–25 (collecting experimental studies). This monolithic view has been widely discredited by federal courts, which recognize—unlike Defendants—that scientific evidence must be considered as a whole, not piecemeal. *See, e.g.*, *In re PPA Prods. Liab. Litig.*, 289 F. Supp. 2d at 1242

[13] Defendants' attempt to criticize experimental studies for "disclaiming" causation is thus just another semantic subterfuge to exclude Plaintiffs' medical experts. Defs.' Mem. at 25.

("Defendants isolate these sources, rather than considering the whole. Non-epidemiological sources are frequently utilized by experts in rendering scientific opinions and, under *Daubert*, should be considered by the court in assessing the reliability of those opinions."). Inviting the Court to deconstruct evidence, rather than examining it as a whole, will lead to one (and only one) result: an abuse of discretion in violation of Rule 702. *See W.R. Grace*, 504 F.3d at 761; *see also Milward v. Acuity Specialty Prods. Grp.*, 639 F.3d 11, 23 (1st Cir. 2011) (district court erred in reasoning the totality of evidence was unreliable because the individual lines of evidence were not sufficient by themselves to support causation); *NutraSweet Co. v. X-L Eng'g Co.*, 227 F.3d 776, 789 (7th Cir. 2000) (concluding that the totality of evidence considered by the expert witness was reliable even though individual pieces of evidence by themselves may be insufficient); *In re Testosterone Replacement Therapy Prod. Liab. Litig.*, 2017 WL 1833173, at *9 (N.D. Ill. May 8, 2017) (holding that experts may rely on individual lines of scientific evidence, which, when combined with other scientific evidence, may support a causal inference).

As explained below, Plaintiffs' medical experts considered the great weight of relevant published data in concluding that there is a causal relationship between Bair Hugger and DJI. Defendants' isolationist approach is anathema to science and law.

V. Dr. Samet Followed Reliable And Relevant Scientific Methods To Support His General Causation Opinion

In his report, Dr. Samet described the guidelines he used, both in his daily practice and for litigation, for making an assessment of causation, citing several recognized authorities such as Glass (2003), Rothman (1976), N.A.S. (2017), U.S. Surg. Gen. (1964),

and Bradford Hill (1965). PX8 (Samet Rpt. at 5, 6, 22 (Table 1)). The particular model Dr. Samet employed in this particular case—the "sufficient component cause framework"—is not only well-accepted among the scientific community but is consistent with other recognized methods for drawing causal inferences.[14] *Id.* at 6–9. All of these methods include the consideration of epidemiologic associations and laboratory evidence. As Sir Bradford Hill put it, "laboratory evidence can enormously strengthen the hypothesis and, indeed, may determine the actual causative agent." PX32 (Bradford Hill, *The Environment and Disease: Association or Causation?* 58 PROC. ROYAL SOC'Y MED. 295, 298 (1965)).

A long line of legal and scientific authority supports Dr. Samet's consideration of multiple sources of evidence for the purpose of drawing causal conclusions in this litigation. *Milward*, 639 F.3d at 25 ("Dr. Smith did not infer causality from this suggestion alone, but rather from the accumulation of multiple scientifically acceptable inferences from different bodies of evidence."); *In re PPA Prods. Liab. Litig.*, 289 F. Supp. 2d at 1243 ("Taking into consideration all of the lines of evidence upon which plaintiffs' experts rely, including the [epidemiologic study] . . . clearly satisfy *Daubert*'s reliability prong.").

Applying the Bradford Hill viewpoints, Dr. Samet began with the epidemiologic evidence from the McGovern study. PX8 (Samet Rpt. at 10–12). He first found that the "temporality" factor was "inherently met because joint replacement and exposure to infectious organisms precede the occurrence of infection, often by months." *Id.* at 16. **None**

[14] Defendants cannot say otherwise given the undisputed fact that their own medical experts admitted that the "sufficient component cause model" employed by Dr. Samet is a valid scientific methodology for drawing causal conclusions. *See* PX2 (Borak Dep. at 67:2–23).

of Defendants' experts dispute this conclusion, which is dispositive here because temporality is the *only* prerequisite to demonstrating causation under the Bradford Hill viewpoints. *See* PX1 (Holford Dep. at 336:12–15) (admitting temporality is met); PX7 (Borak Rpt. at 5) ("[C]oncern about temporality is not an issue here because, by definition, use of warming devices during surgery precede the development of post-operative SSI."); REFERENCE MANUAL at 601 (noting that temporality must exist to draw causal inferences).

Dr. Samet then concluded that the "strength of association" factor strongly favored an inference of causation given the McGovern study's reported odds ratio of 3.8. *See* PX8 (Samet Rpt. at 11, 16); *see also* PX13 (Samet Dep. at 62:6-63:7; 246:12-247:3). Indeed, "the higher the relative risk, the greater the likelihood that the relationship is causal." REFERENCE MANUAL at 602. Furthermore, because the reported association was statistically significant, Dr. Samet found that the association was highly likely due to a "real" effect as opposed to one caused by "bias or chance." PX8 (Samet Rpt. at 11); *Matrixx Initiatives, Inc. v. Siracusano*, 563 U.S. 27, 40 (2011) ("statistical significance is [not] the only reliable indicator of causation"); Plfs.' Mem. to Exclude Holford at 12–17. Defendants' experts volunteered similar information. **Professor Holford, for instance, agreed that "an odds ratio of less than two can show causation" but that was no issue here because even his revised estimate of the odds ratio of exceeded 2.0 when he controlled for hypothetical confounders.** *See* PX1 (Holford Dep. at 345:22-346:25).

Dr. Samet next considered whether potential confounding factors impacted the findings of the McGovern study. *Id.* at 11–12. He acknowledged that McGovern et al. had commented on the *potential* limitations of the study, namely changes in antibiotic

prophylaxis and thromboprophylaxis, but Dr. Samet certainly did not ignore these potential confounders as Defendants suggest. *See id.* Based on his review of the peer-reviewed literature and the data and testimony from the authors of the McGovern study, **Dr. Samet found that antibiotics and anti-thrombotic drugs have *not* been shown to increase the risk of DJI.** *Id.* Nor in his opinion is there any scientifically convincing evidence that these drugs or changes in SSI bundles influenced the rate of DJI in the McGovern study. *See id.*

With the first two Bradford Hill criteria met, Dr. Samet considered the final two factors of "consistency" and "coherency" (also known as "biological plausibility"). He found strong "consistency of the findings of studies addressing the effect of the Bair Hugger device on particle counts at the surgical site." PX8 (Samet Rpt. at 16). He also found "coherency" across multiple streams of scientific evidence, creating a "well-supported picture of mechanisms by which the Bair Hugger device increase risk for deep joint infections." *Id.* Defendants' experts agreed with these conclusions albeit for different reasons. PX1 (Holford Dep. at 352:19) ("[N]ow there is a consistency of two."); *see id.* 365:3-372:3 (agreeing that "mechanistic studies" show "biological plausibility" and studies regarding similar devices "coincide[] with a lot of the concern" about Bair Hugger).

Indeed, Dr. Elghobashi's computational fluid dynamics analysis along with a plethora of published papers discussing the mechanisms for causation—both positive and negative—clearly establish consistency and coherency across multiple lines of scientific evidence. *See generally* Pls.' Opp. to Defs.' Mot. to Exclude Engineering Experts. These studies measured the increase in particle counts, microbes, and infection rates at the surgical site after using Bair Hugger. *See* PX8 (Samet Rpt. at 12–16). **Dr. Samet found**

that these studies supported the conclusion that Bair Hugger increases the number microbes at the surgical site and that such an increase increases the risk of DJI. *Id.*

For example, Darouiche et al. recently published the results of a randomized clinical trial—the "gold standard" as Defendants call it—which demonstrated that as the number of particles increase at the sterile surgical site, so too do the number of contaminants, which in turn increases the risk of DJI among orthopedic patients. *See* PX17 (Darouiche 2017 at 14); *see also* PX33 (Stocks 2010) (concluding that particles are a proxy for bacteria); PX34 (Raval 2012) ("[R]educed airborne particulates appear to correlate with a decreased risk of nosocomial infection in high-risk patient populations."); *cf.* PX35 (PJI Consensus Statement at 115–16) (reaching a strong consensus that reducing the amount of airborne particles in operating rooms will reduce amount of bacteria and thus the risk of infection).[15]

It is also undisputed that every experimental study to date, *including those conducted by Defendants*, has found that Bair Hugger increases the number of particles at the surgical site. *See, e.g.*, PX36 (Sessler 2011) (study was funded, conducted, and edited by 3M and found Bair Hugger increases particles at surgical site); [redacted] *see also* PX14 (McGovern 2011) (statistically significant increase); PX38 (Legg 2012) (same); PX39 (Legg 2013) (same); PX40 (Belani 2013) (same); PX41 (Wood 2014) (collecting studies). The Court need only turn to the sworn testimony of Defendants' corporate representative

[15] On cross-examination, Professor Holford agreed to this very same chain of causation, effectively supporting a showing of general causation. *See* PX1 (Holford Dep. at 75:3–18).

to rest soundly assured that [redacted][16] [redacted]

Other independent studies have further found that Bair Hugger increases both **particles *and* bacteria** at the surgical site. *See, e.g.*, PX43 (Moretti 2009); PX13 (Samet Dep. at 315:11–25) (citing Moretti 2009); PX41 (Wood 2014) ("Moretti et al. found an increased bacterial load at the surgical site when FAW was used."); *see also* PX44 (Tumia 2002) (reporting "higher" bacteria from Bair Hugger).[17] For these reasons, it's no wonder the CDC has instructed that "[n]othing that blows air should be in an operating theatre." PX45 (CDC 2015 Proceedings at 27); *see also* PX46 (CDC 2016 Proceedings at 29) ("devices that blow air should probably not be situated in high-risk locations"); *cf.* PX47 (FDA Safety Alert) (instructing doctors to direct "exhaust away from the surgical field to mitigate the risk of aerosolizing [bacteria] into the sterile field and exposing the patient").

Nor were the vast majority of these studies "orchestrated" by Dr. Augustine or his corporate entities.[18] Defs.' Mem. at 14. In fact, Defendants have cited the Moretti study for

[16] [redacted]

[17] On reply, Defendants may argue that Moretti et al. did not find an increased risk of DJI among patients exposed to Bair Hugger. This is nonsense. For the same reasons that Professor Holford admitted that his calculations were not only underpowered but variable, the same problems defeat Moretti's analysis of whether only 20 patients developed a DJI. [redacted]

[18] To the extent Defendants argue on reply that these peer-reviewed studies hold no weight because of their supposed connection to Dr. Augustine, Defendants apparently forget that

nearly a decade despite its results. *See* PX43 (Moretti 2009) (finding that Bair Hugger significantly increases bacteria at the surgical site). The Legg 2013 study, moreover, found that "convection currents [from the Bair Hugger] **increased particle concentration 1000-fold** by drawing potentially contaminated particles from below the operating table into the surgical site." *See* PX39 (Legg 2013). The study makes clear that "[n]o benefits in any form [were] received from a commercial party directly or indirectly relating to the subject of this article." *Id.* Nor were any of the study authors employed by Dr. Augustine or his corporate entities. Dr. Legg testified that he drafted the paper and conducted the scientific research separate and apart from Dr. Augustine. PX50 (Legg Dep. at 65:11–25, 81:18–25).

The Legg 2012 study similarly found that Bair Hugger significantly increased the number of particles in the surgical field, "which raises concern as bacteria are known to require particles for transport." PX38 (Legg 2012). There, too, the authors had no personal or professional relationship with Dr. Augustine. Disclaiming any such connection, the authors of the study testified under oath that they independently designed, conducted, and published their experiment and related research. PX50 (Legg Dep at 39:22–24, 42:3–16).

The same conclusion holds true for a slew of other experimental studies that predate Dr. Augustine's departure from the company. *See, e.g.*, PX52 (Avidan 1997) (concluding that Bair Hugger is a "potential source of nosocomial infection"); PX53 (Bernards 2004) (finding bacteria inside Bair Hugger was linked to outbreak of nosocomial infections); PX54 (Gjolaj 2009) (declaring that Bair Hugger "may blow contaminated air"); *see also*

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the number one study that they have relied on inside and outside the courtroom to "prove" the safety of the Bair Hugger was also funded by Dr. Augustine. *See* PX51 (Zink 1993).

PX55 (Beavers 2014) (concluding that Bair Hugger is a "reservoir of infection"). **Accordingly, even without considering Dr. Elghobashi's robust CFD analysis, all of these independent studies support Dr. Samet's opinion that Bair Hugger causes DJI.**

Dr. Samet also refused to shy away from "contrary" evidence. He considered the studies routinely cited by Defendants and explained why each of those studies was not scientifically persuasive evidence that Bair Hugger does not increase the risk of DJI. *See id.* at 13. **He reasoned, for example, that all those studies are plagued by small sample sizes and therefore have insufficient statistical power to measure causal effect.** *Id.* As numerous scientific authorities similarly point out, indicators of risk "can easily be masked by poor exposure classifications" and small populations that yield poor power. PX4 (Clapp at 209). Indeed, "most forces operate to lower the observed risks, not raise them." *See id.*

Based on his consideration of multiple and competing lines of evidence, Dr. Samet faithfully followed an accepted methodology, found that all of the applicable Bradford Hill criteria were satisfied, and therefore reliably concluded that Bair Hugger "constitute[s] a substantial contributing cause" of DJI. *See* PX8 (Samet Rpt. at 17). He not only explained precisely how he went about arriving at his conclusions, but he cited a multitude of published, peer-reviewed research to show that he followed the same method that he and his colleagues use in their field of research. *See, e.g.*, *McClellan*, 710 F. Supp. 2d at 1102.

Dr. Samet's thorough analysis stands in stark contrast to Defendants' medical experts, who cite no legitimate authority for manufacturing data by "remixing" and "slicing and dicing" data from different studies to reengineer the findings of a published study. *See* Pls.' Mem. to Exclude Holford; *see also* Pls. Mem. to Exclude Borak. Defendants therefore

cannot object to the methodology that Dr. Samet employed in developing his opinion on general causation. Their criticism of him is largely limited to his consideration of the McGovern study even though they do not contest that "epidemiological evidence is not absolutely necessary to support general causation expert opinions," Defs.' Mem. at 24, and even though their medical experts concede that the only required Hill factor is readily met here. *See* PX1 (Holford Dep. at 336:12-15) (admitting temporality); PX7 (Borak Rpt. at 5).

A. Statements by Medical Professionals Who Have Not Conducted Causation Analyses Go to Weight, Not Admissibility of Testimony.

Although Defendants regurgitate cherry-picked snippets from "medical professionals and medical device regulators" as "independent" evidence "disclaim[ing] any finding of causation," none of these statements—independently or collectively—justify excluding Dr. Samet's opinion regarding general causation. Defs.' Mem. at 27–28.

Above all these statements, Defendants tout the FDA's recent "Safety Alert." *Id.* at 1, 28. Close inspection of the one-page letter, however, abrogates its import. **Like Dr. Borak's expert report, the letter does not even address the outcome of interest in this litigation—deep joint infection**; **rather, it addresses a broader category of infections called "surgical site infections."** DX1 at 1–2; *see* Pls.' Mem. to Exclude Borak at 8–10.

The difference is not a matter of semantics but of science and fit. Since surgical site infections are "either incisional or organ/space," they often involve skin and subcutaneous tissue. PX56 (Jarvis Rpt. at 6–7). Deep joint infections, on the other hand, occur beneath the skin and tissue. *See id.* Given that distinction, "large numbers of micro-organisms [are] required to cause [surgical site] infections, [whereas] as few as 1-10 [colony-forming units]

are required to cause [deep joint infections]." *Id.* at 16. Not to mention that Plaintiffs are especially susceptible to infection because "[a]ny indwelling medical device or prosthetic implant has the potential to become colonized by organisms embedded in biofilm." *See id.*

Though the etiology of DJI is different from surgical site infection, the FDA's letter conflates the two and thus holds limited if any scientific value here. Defendants' experts admitted so when cross-examined on the "fit" of surgical site infections to the injuries at issue in this litigation. *See, e.g.*, PX1 (Holford Dep. at 305:3-306:10) (admitting the two types of infections were "not the same thing"); *see also* PX56 (Jarvis Rpt. at 8) ("One cannot generalize from one surgical procedure (i.e., cardiac or general surgery) to another (orthopedic or specifically to total knee arthroplasty or total hip arthroplasty procedures).").

The FDA, moreover, did not even attempt to review the same evidence that is now before the Court. Unlike Plaintiffs' medical experts, the FDA only relied on "data available" from "several" public sources. It thus did not consider the full gamut of mechanistic evidence, including but not limited to the alarming results of Dr. Elghobashi's analysis. Nor did the FDA consider the sworn deposition testimony that Plaintiffs' have secured in this litigation when it surmised that forced-air warming systems "decrease[] [the] risk of infection." *See* Defs.' Mem. at 1 (citing DX1). While there does exist an outdated and underpowered study that concluded that forced-air warming might reduce the risk of *surgical site infections* in *colorectal surgeries*, that findings does not fit and has nothing to do with orthopedic surgery, where it only takes a small inoculum of bacteria to cause DJI. *See* PX57 (Kurz 1996); [redacted]

[redacted]

What's more, when the first-author of the study was crossed-examined in this case, she swore that under **"today's scientific standards, there is *no reliable evidence* that supports that maintaining normothermia reduces the incidence of infection."** PX59 (Kurz Dep. at 179:16–20.) (emphasis added). The FDA did not consider that admission; nor did it apparently review the recent results of the Brown study, which concluded that "[i]ntraoperative hypothermia [is] not significantly associated with SSI." PX60 (M. Brown, et al. *Intraoperative Hypothermia and Surgical Site Infection in Patients with Class I Clean Wounds: A Case-Control Study*, J. AM. COLL. SURG. 224, 160–171 (February 2017)).

On the face of it, the FDA's one-page letter falls far short of satisfying any rigorous causation methodology, such as the one that Dr. Samet followed in this litigation. *See, e.g.*, PX4 (Clapp at 199) ("Epidemiologists concerned with the causes that contribute to human [disease] routinely use the [Bradford Hill viewpoints to draw] deductions about causation from all the available relevant principles, data, information, and observations."). This Court should therefore follow the overwhelming majority of courts in concluding that the FDA's one-page letter holds little if any weight when it comes to the admissibility of differing expert opinions on scientific issues. *See, e.g.*, *In re Testosterone Replacement Therapy Prod. Liab. Litig.*, 2017 WL 1833173, at *13 (N.D. Ill. May 8, 2017) ("[A]lthough the FDA may have a different interpretation of the studies relied upon by plaintiffs' experts, it is left to the trier of fact, not the reviewing court, to decide how to weigh the competing expert testimony."); *In re Celexa & Lexapro Prod. Liab. Litig.*, 927 F. Supp. 2d at 765 (refusing to exclude expert testimony in view of contrary findings and statements from the FDA).

The remaining pamphlets and articles cited by Defendants are superficial and flawed since they are not peer-reviewed[19] or not what Defendants say they are, *i.e.*, "independent" reviews. *See* Defs.' Mem. at 26. Defendants cite a 2013 article by the ECRI Institute, for instance, which tersely concluded that the "currently available evidence [did not] justif[y] discontinuing the use of FAW during surgery." DX15. But Defendants do not mention that [redacted] Defendants also never mention that [redacted] **Defendants even fail to mention that** [redacted]

If that were not enough to question the effect of Defendants' tactics, they fail to mention ECRI's more recent recommendations. On May 1, 2017, ECRI warned that all "forced-air warming units should have HEPA-grade or better air filters to reduce the risk that airborne dust, bacteria, and mold will be blown onto the patient or into wounds." PX64 (ECRI Article). Defendants not only fail to mention that Bair Hugger does not have—and has never had—a HEPA-grade or better filter, **but they omit the fact that they waited until this litigation to** [redacted]

[19] These sources include but are not limited to the article published by the ECRI Institute (DX15), the FDA's one-page "Safety Alert" (DX1), and a two-page newsletter (DX22).

[redacted]

To the extent Defendants actually cite peer-reviewed literature, none of these sources *disprove* the conclusions of Plaintiffs' experts. Just the opposite is true. Consistent with Dr. Elghobashi's conclusions, for example, the "independent review" by Sikka and Prielipp recognizes that "forced air warming can impact laminar flow" in orthopedic surgeries. *See* Defs.' Mem. at 27. Though the authors noted that "any actual clinical impact on surgical site infections must be considered unproven" as of 2014, they did not address the outcome of interest in this case (deep joint infections), let alone disprove the opinions of Plaintiffs' experts based on the same totality of information. *Id.* The same holds true with respect to statements from the 2013 Consensus Meeting of Periprosthetic Joint Infection, which was not only funded by 3M as its "platinum sponsor" but recognized the "theoretical risk posed" by forced-air warming devices given the connection between airborne particular matter and DJI. *See* DX18; PX35 (PJI Consensus Statement at 115–16) (recognizing that particles are a proxy for bacteria and lead to DJI). Moreover, all the statements relied upon by Defendants contradict the opinions of the scientists who carefully evaluated and peer-reviewed the McGovern study itself. As one peer-reviewer put it: The McGovern study "demonstrates that there were actual changes in infection rates which would fit well with the experimental data and therefore support the contention that there is a serious issue to be addressed with [FAW] devices." PX16 (McGovern Dep. at 375:9–14).

In sum, none of these publications reviewed the Hill criteria or any other scientific method for drawing causal conclusions; and none of them considered the same information

that Plaintiffs' medical experts have relied on this litigation, including but not limited to the deposition testimony of third-party scientists, Defendants' employees, and expert witnesses; internal documents, data, and reports produced by third-party scientists and Defendants; randomized controlled trials proving the relationship between particles, bacteria, and DJI; the universe of epidemiologic and experimental research regarding the risks of using Bair Hugger in orthopedic procedures; and the reports of Plaintiffs' experts.

Compare Dr. Samet, who reviewed **196 independent sources of evidence**—ranging from peer-reviewed literature, to Defendants' internal documents, to the deposition testimony of party and non-party witnesses—in determining whether Bair Hugger can cause DJI, *see* PX66 (Samet Ex. C), **to the one-page letter from the FDA that failed to cite a single scientific source**.[20] DX1; *see also* DX15 (ECRI Article – 15 sources); DX16 (AORN Article – 20 sources); DX18 (PJI Consensus – 9 sources); DX22 (Duke Article – 8 sources); DX23 (Allen Article – 12 sources re increased risk; others on unrelated topics).

This fact alone distinguishes the general causation opinions of Dr. Samet from the specific causation opinions at issue in *Glastetter*, which were not only "*ipse dixit*" but contradicted the "leading treatise" on the relevant subject matter. *See Glastetter*, 252 F.3d at 990; *cf.* Defs.' Mem. at 26 (citing *Glastetter* for an inapposite proposition). Unlike in

[20] Consistent with this approach, Dr. Samet reviewed a recent article by Dr. Augustine reporting a strong association between Bair Hugger and DJI. *See* Defs. Mem. at 20 n.6. Although Defendants' experts conceded that the statistics reported in that article were less variable and thus more reliable than the calculations in their reports, *see* PX1 (Holford Dep. at 335:2–8), Dr. Samet did not rely on the Augustine article in his expert report. *See generally* PX8 (Samet Rpt.); PX66–67 (Samet Exs. B, C). Nor could he have done so since the article was published months *after* Plaintiffs' medical experts submitted their reports.

Glastetter, moreover, all of the experimental evidence coheres, not contradicts, Dr. Samet's opinions; not to mention that Defendants' internal documents [redacted]

In the final analysis, Defendants cannot cite a single peer-reviewed study that contradicts the statistically significant finding of DJI risk associated with Bair Hugger; and even if they could, such a study would go only to the weight of the evidence, not the admissibility of expert testimony under Rule 702. *See, e.g.*, *Johnson*, 754 F.3d at 562; *Hill*, 858 F.3d at 486. That is especially true where, as here, Defendants have not only admitted [redacted]

Accordingly, Defendants' motion to exclude Dr. Samet's opinions should be denied.

VI. Dr. Jarvis's Testimony Is Admissible Under *Daubert*

A. Dr. Jarvis Applied the Same Methodology He Developed at the CDC.

Dr. Jarvis worked at the CDC for 23 years, investigating the causes of infection outbreaks throughout the world. His report explains the "gold standard" methodology he developed at the CDC to conduct investigations: "[M]y team and I developed a systematic methodology to investigate infectious disease outbreaks or other complications associated with healthcare delivery. This method centered around a systematic epidemiological-based approach to the investigation of infectious disease outbreaks." PX56 (Jarvis Rpt. at 2). Dr. Jarvis used the identical approach in researching and preparing his expert report. *Id.* at 3.

He reached his opinions by employing the same rigor and scientific steps he and other colleagues in his field would use in analyzing what role, if any, Bair Hugger plays in causing DJI. His analysis is therefore trustworthy. *See Kumho Tire Co.*, 526 U.S. at 152.

Defendants nevertheless assert that Dr. Jarvis's testimony stands "in stark contrast" to the rigor of his work at the CDC. They specifically focus on a single sentence in his report which notes that "[e]xogenous sources account for the majority of SSIs." PX56 (Jarvis Rpt. at 5.) Defendants assert that this statement contradicts the 1999 CDC Surgical Site Infection Prevention Guideline that Dr. Jarvis co-authored. Defs.' Mem. at 29. The Guidelines state: "For most SSIs, the source of pathogens is the endogenous flora of the patient's skin, mucous membranes, or hollow viscera." *See* PX69 (1999 Guideline at 253).

This argument ignores Dr. Jarvis's sworn testimony, which clearly dispels Defendants' false accusation. At his deposition, Dr. Jarvis explained the distinction, pointing out that the entire 1999 CDC Guideline addressed both endogenous and exogenous sources of bacteria that cause infections. *See* PX70 (Jarvis Dep. at 155:13–19).

Dr. Jarvis also explained that surgical environments, methods, and practices have changed over the last two decades. The 1999 CDC Guidelines cited a study from 1968 in support of the reference to "endogenous" sources. *See* PX70 (Jarvis Dep. at 155:13-156:24; 154:23, 158:5–7, 164:7–9). Although the 1999 CDC Guideline was correct at the time it was written, it was based on limited information. Dr. Jarvis's opinions take into account the evolution in surgical environments, methods, and practices since the 1999 CDC Guidelines were published, as well as the differences in orthopedic versus general surgery. The Guidelines relate to surgical site infections *of any kind*, not to orthopedic implant

surgeries—a critical distinction for purposes of this litigation. *Id.* at 51:16-53:9. The alleged inconsistency in Dr. Jarvis's report and the 1999 CDC Guidelines is just another red-herring. Defendants cite no evidence to support their bare-bones assertion, and if such evidence existed, it would go to weight, not admissibility. *See Kuhn*, 686 F.3d at 627–28.

Defendants next maintain that Dr. Jarvis's "heavy" reliance on the published and peer-reviewed McGovern study would not meet his scrutiny were he still employed at the CDC. *See* Defs.' Mem. at 31–33. This meritless argument ignores his expert report, his testimony, and well-accepted methods all epidemiologists use in assessing causation:

> Depending upon a scientist's judgment of external validity of a particular study, he or she may decide that an individual piece [of the evidence puzzle] forms a large and central part of the picture, is just a small piece on the periphery of the picture, or not part of the picture at all. In addition, a scientist's experience, expertise and basic judgment are involved.
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> ***
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> In sum, scientists may, and often do, disagree about which pieces are internally and externally valid, and disagree about just how to assemble the internally and externally valid puzzle pieces. [W]hat scientists do not disagree about, though, is that they routinely select pieces and assemble such pictures and call the end product of this process an explanation.

PX4 (Clapp at 212).

Dr. Jarvis weighed the McGovern study along with dozens of other relevant sources of scientific evidence from the published literature, applying his own highly relevant multi-disciplinary experience, expertise, and judgment in reaching his opinion. *See* PX70 (Jarvis Dep. at 189:22-190:14); *see generally* PX56 (Jarvis Rpt.). Defendants may disagree with

his conclusions and the weight he gives particular pieces of evidence, but the correctness of his opinion is for the jury to decide. *See, e.g.*, *Hose*, 70 F.3d at 976. Because Dr. Jarvis's testimony is based on solid methodological and infection control principles, it is admissible and easily falls within "the range where experts might reasonably differ." *Johnson*, 754 F.3d at 564. The Court should thus deny Defendants' motion to exclude Dr. Jarvis. *See id.*

VII. Dr. Stonnington's Methodology Is Reliable And His Unique Clinical Experience Will Assist The Jury

A. Dr. Stonnington's Opinion Is Based on His Experience with the Device.

As an orthopedist and Chair of Surgery at a busy hospital, Dr. Stonnington has years of experience with the use of Bair Hugger and other warming devices during hip and knee implant surgeries. Based on his clinical experience and the published literature, Dr. Stonnington no longer uses Bair Hugger in his medical practice focused on joint surgeries, and he recommends that his colleagues use alternative forms of patient warming. *See* PX11 (Stonnington Rpt. at 8). His opinions here are thus entirely consistent with this day to day clinical decision-making to protect the safety of his patients. Given the alignment of his clinical practice and his opinions in this case, tied to his qualifications, experience, and background, Dr. Stonnington's testimony is reliable and can assist jurors in understanding the nuances of operating room practice, particularly in the context of orthopedic devices.

Defendants' chief complaint against Dr. Stonnington is that his clinical observations amount to "case reports," which they claim are inherently unreliable because they lack a control group, do not explain the mechanism of causation, and do not account for other potential causes. *See* Defs.' Mem. at 33–34. This criticism is misleading because it fails to

appreciate the proper role of case reports and clinical experience in making causal inferences, and it simply leaves out other important elements of Dr. Stonnington's opinion. *See* REFERENCE MANUAL at 714 (identifying "case reports" as evidence physicians "often use" to "support causation"); *In re Accutane Prod. Liab.*, 2007 WL 2340496, at *1 (M.D. Fla. Aug. 15, 2007) ("Case reports may be a factor in determining causation if used in a manner that is scientifically reliable."); *cf. Matrixx Initiatives, Inc.*, 563 U.S. at 40 (concluding that adverse event reports were relevant evidence for determining causation).

In addition to case reports, Dr. Stonnington bases his opinion on his assessment of the medical literature, which he describes in detail in his report. *See* PX11 (Stonnington Rpt. at 3–7). Dr. Stonnington testified repeatedly that he does not rely on any single study or case series, but rather on all the evidence at his disposal, including his experience and training as an orthopedic surgeon, his many years of clinical practice, his discussions with colleagues and related investigations, his review of relevant literature and other evidence, and his clinical experience with Bair Hugger. *See, e.g.*, PX71 (Stonnington Dep. at 19:13–20, 25:2-26:22, 118:23-120:19, 126:16-127:9, 173:11–17, 271:24-273:4, 274:2-275:7).

It is well settled in this Circuit and elsewhere that medical experts need not base their opinions on epidemiological research to opine on causation. *See, e.g.*, *Bonner*, 259 F.3d at 929; *Glastetter*, 252 F.3d at 992. As one federal court put it, "[n]on-epidemiological sources are frequently utilized by experts in rendering scientific opinions and, under *Daubert*, should be considered by the court in assessing the reliability of those opinions." *See, e.g., In re Phenylpropanolamine (PPA) Prods. Liab. Litig.*, 289 F. Supp. 2d at 1242.

Courts admit medical testimony based in part on relevant clinical experience because it provides useful information about causation. *See Huggins v. Stryker Corp.*, 932 F. Supp. 2d 972, 991–92 (D. Minn. 2013); *see also Kloss*)*v. Wal-Mart Stores, Inc.*, 2013 WL 268936, at *7–9 (D. Minn. Jan. 24, 2013) (admitting causation testimony of physicians based on their clinical experience even though their opinions were based on anecdotal evidence, not well-documented studies); *Donner v. Alcoa Inc.*, 2014 WL 12600281, at * 3 (W.D. Mo. Dec. 19, 2014) (admitting causation testimony of physician who relied on case reports and published articles); *cf. Marmo v. Tyson Fresh Meats, Inc.*, 457 F.3d 748, 758 (8th Cir. 2006) (concluding that medical expert had only interviewed, not examined, the plaintiff). Whether case reports alone are sufficient to establish causation is not even at issue here because they fit within the totality of the scientific evidence now before the Court, including the epidemiologic and experimental studies that physicians routinely use to draw causal inferences. *See, e.g.*, *In re PPA Prods. Liab. Litig.*, 289 F. Supp. 2d at 1242.

Moreover, Dr. Stonnington's observations are distinguishable from individual case reports because they derive from his intimate familiarity with how physicians actually use Bair Hugger in the surgical procedures at issue in this litigation. This unique experience is informed by his regular review of relevant literature. Besides treating hip and knee implant patients for many years, Dr. Stonnington also oversees the operating room as Chief of Surgery. His clinical observations and judgments concerning patient safety—and specifically infection control—are both reliable and relevant. *See, e.g.*, *Huggins*, 932 F. Supp. 2d at 991–92; *Kloss*, 2013 WL 268936, at *7–9; *Donner*, 2014 WL12600281, at *3.

Defendants also falsely maintain that Dr. Stonnington did not disclose "any data whatsoever" on the infection rates in his clinical practice during and after discontinuation of Bair Hugger. Defs.' Mem. at 33. In his deposition, however, Dr. Stonnington clearly described his clinical experience with DJI rates before and after Bair Hugger as follows:

> So when you average all those years from 2005 to 2014, the average – the infection rate was above zero. When you look at 2015 and beyond, it's zero. What I changed was getting Bair Hugger out of the operating room. Everything else stayed the same. Antibiotic prophylaxis, everything.

PX71 (Stonnington Dep. at 64:13–18). Thus, Defendants' argument that Dr. Stonnington disclosed no data "whatsoever" on DJI rates during his clinical practice is simply wrong.

Dr. Stonnington's "challenge/dechallenge" experience with Bair Hugger is also reliable and relevant information for purposes of establishing causation. The Eighth Circuit has recognized as much in stating that such data are "substantially more valuable than run-of-the-mill case reports." *See Glastetter*, 252 F.3d at 990. Dr. Stonnington's experience with challenge/dechallenge data derives from a large number of hip and knee implant procedures; on average, he performs over 250 total joint replacement surgeries per year, over the course of his nearly 17 year career. PX11 (Stonnington Rpt. at 1). These first-hand observations from a veteran surgeon, combined with relevant epidemiological and experimental literature, weigh strongly in favor of admissibility under the permissive standards of *Daubert* and Rule 702. *See Marmo*, 457 F.3d at 758 ("Courts should resolve doubts regarding the usefulness of an expert's testimony in favor of admissibility.").

B. Dr. Stonnington Took Action After Learning Bair Hugger Caused DJI.

Defendants finally complain that Dr. Stonnington did not report adverse events regarding infections to the FDA, *see* Defs.' Mem. at 34, even though Dr. Stonnington took action in his clinical practice to notify other authorities and to protect his patients from unnecessary DJI risk. PX72 (Issa Dep. Ex. 275). Dr. Stonnington even stopped using Bair Hugger altogether for a period of time. PX71 (Stonnington Dep. at 51:9–20). The hospital sent this information to Defendant 3M only to receive a boilerplate response citing the ECRI article and summarily dismissing its concerns. PX73 (Stonnington Dep. Ex. 8).

While Dr. Stonnington is not required to submit Adverse Event Reports ("AER's") to the FDA, Defendants *are* required to do so. *See* 21 C.F.R. § 803. After 3M received emails from Dr. Stonnington, Defendants did not submit this information to the FDA; nor did it undertake any investigation or attempt to gather the information necessary to report adverse events to the FDA as required by federal law. *Id*. Defendants' internal documents show that [redacted] In fact, hundreds if not thousands of doctors and hospitals [redacted] In the words of the Bone & Joint Center: [redacted]

For example, one doctor informed Defendants that [redacted]

[redacted] *Id.* Still other surgeons have expressed [redacted] Nor can Defendants deny that after a patient [redacted] Given these concerns, [redacted]

At bottom, Defendants' attack on Dr. Stonnington is cynical at best and fails to inform the *Daubert* inquiry at worst. It was incumbent on 3M, not Dr. Stonnington, to alert the FDA to the foregoing information. Dr. Stonnington's testimony meets the threshold for reliability and fit under *Daubert*. And given his extensive clinical experience with Bair Hugger, his testimony will assist the jury. The motion to exclude him should be denied.

VIII. The General Causation Opinions Of Plaintiffs' Medical Experts Are Admissible Under Minnesota Law As Well

In Minnesota, if expert opinion or evidence involves novel scientific theories or techniques, the proponent of the evidence must show that "the underlying scientific evidence is generally accepted in the relevant scientific community." Minn. R. Evid. 702. Under this two-pronged *Frye-Mack* standard, opinion testimony is admissible if: (1) the technique or theory used by the expert is generally accepted in the relevant scientific community; and (2) if the foundation for the expert's opinion is reliable. *Goeb v. Tharaldson*, 615 N.W.2d 800, 814 (Minn. 2000). This standard does not apply to opinions that are neither new nor novel. *State v. Jensen*, 482 N.W.2d 238, 239 (Minn. App. 1992).

A. Minnesota Law Does Not Prohibit the Opinion Testimony of Plaintiffs' Medical Experts Because Plaintiffs' Theory of Causation Is Not Novel.

In many medical device cases, experts do not have the benefit of epidemiologic evidence. Not so here. The published and peer-reviewed McGovern study shows that Bair Hugger significantly increases the risk of DJI in orthopedic patients. This association is supported by numerous experimental and clinical studies demonstrating two mechanisms by which Bair Hugger moves particles and bacteria to (rather than away from) the surgical site—one by emitting contaminants directly from the device to the sterile surgical site, and the other through disrupting operating room airflow and thereby moving contaminants toward the surgical field. *See, e.g.*, PX8 (Samet Rpt. at 14) (describing two mechanisms).

Plaintiffs' medical experts not only rely on both lines of epidemiologic and experimental evidence, but they also use well-established scientific methods to reach their causal determinations. As a result, their opinions are not "novel" or otherwise subject to exclusion simply because no single piece of research simultaneously studied epidemiologic associations and either one of the mechanisms by which Bair Hugger causes DJI. Furthermore, it is generally accepted that aerosolization of bacteria from machines blowing air in the operating room can cause infections. *See* PX45 (CDC 2015 Proceedings at 27); PX17 (Darouiche 2017) (finding an association between particles, bacteria, and DJI); *cf.* PX47 (FDA Safety Communication) (direct "exhaust away from the surgical field to mitigate the risk of aerosolizing [bacteria] into the sterile field and exposing the patient").

Because the causation testimony of Plaintiffs' medical experts falls well outside the scope of *Frye-Mack*, Defendants' motion to exclude Plaintiffs' experts should be denied.

B. The Opinions of Plaintiffs' Experts Satisfy *Frye-Mack* Because the Methods They Used to Reach their Conclusions Are Generally Accepted.

Under the "general acceptance" prong of the *Frye-Mack* standard, courts consider whether "novel scientific *evidence*" is generally accepted in the relevant scientific community. *See State v. MacLennan*, 702 N.W.2d 219, 230 (Minn. 2005) (emphasis added). The inquiry of "general acceptance" thus does not depend on whether there is widespread consensus that an expert's *conclusions* are correct, but rather whether there is general acceptance that the expert's *methodology* or "technique" is correct. *State v. Taylor*, 656 N.W.2d 885, 891 (Minn. 2003) (emphasis added). Were it any other way, no causation expert would ever be allowed to testify. The Florida Supreme Court makes this very point:

> Under *Frye*, the inquiry must focus only on the general acceptance of the scientific principles and methodologies upon which an expert relies in rendering his or her opinion. Certainly the opinion of the testifying expert need not be generally accepted as well. Otherwise, the utility of expert testimony would be entirely erased, and "opinion" testimony would not be opinion at all—it would simply be the recitation of recognized scientific principles to the fact finder. . . . We reaffirm our dedication to the principle that once the *Frye* test is satisfied through proof of general acceptance of the ***basis of an opinion***, the expert's opinions are to be evaluated by the finder of fact and are properly assessed as a matter of weight, not admissibility.

Marsh v. Valyou, 977 So.2d 543, 549 (Fla. 2007) (citations omitted) (emphasis added); *see also Anderson v. Akzo Nobel Coatings, Inc.*, 260 P.3d 857, 866 (Wash. 2011) ("If we were to accept [defendant's] argument and require 'general acceptance' of each discrete and evermore specific part of an expert opinion [under *Frye*], virtually all opinions based upon scientific data could be argued to be within some part of the scientific twilight zone.").

As described above, Plaintiffs' medical experts followed scientific methodologies that are routinely used by their peers. The Bradford Hill criteria is, without question, generally accepted by epidemiologists in determining whether an association is causally related to injury. *See* REFERENCE MANUAL at 599–600. Indeed, courts routinely embraced these criteria as a reliable methodology for inferring causation, just as Plaintiffs' medical experts have used them here. *See id.* at 599 n.141; *see also In re Viagra Prods. Liab. Litig.*, 572 F. Supp. 2d at 1081. While Defendants dispute how Plaintiffs' experts have interpreted the evidence, those disagreements go to the weight of their testimony, not its admissibility. *See, e.g.*, *Sentinal Mgmt. Co. v. Aetna Cas. & Sur. Co.*, 615 N.W.2d 819, 825 (Minn. 2000).

The testimony of Plaintiffs' medical experts also satisfies the "reliability" prong of the *Frye-Mack* standard. Drs. Samet, Jarvis, and Stonnington all base their causal determinations on a large volume of published, peer reviewed literature—including epidemiologic studies and mechanistic data—to which they applied their specialized knowledge and experience. The experts also explained why alternative factors did not confound the findings in the McGovern study and why "negative" studies did not affect their opinions. Under Minnesota law, Defendants' criticisms of any attendant deficiencies are for the jury, not the Court, to decide. *E.g.*, *Sentinal Mgmt. Co.*, 615 N.W.2d. at 824–25.

In sum, the testimony of Plaintiffs' experts is reliable, relevant, and thus admissible. Defendants' motion to exclude these opinions should be denied pursuant to Minnesota law.

CONCLUSION

For all of the reasons stated above, Plaintiffs respectfully request that the Court deny Defendants' Motion to Exclude Plaintiffs' General Causation Medical Experts.

Respectfully submitted,

Dated: October 3, 2017

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